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THE PRESSORECEPTIVE MECHANISMS FOR THE REGULATION OF HEART RATE, VASOMOTOR TONE, BLOOD PRESSURE AND BLOOD SUPPLY

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FOR many years physiologists have been occupied with the study of the mechanisms which regulate the heart rate, the vasomotor tone and the blood pressure. A precise and detailed knowledge of the physiologic regulation of endovascular pressure is the key, not only to the physiology of normal circulation, but also to the pathogenesis of chronic arterial hypertension.

It is known that the pressure in the large arteries is controlled principally by the activity of the heart, the cardiac rate, the cardiac output, the circulating blood volume and, mainly, by the peripheral vascular resistance, the vasomotor tone.

Although the arterial pressure is dependent upon these factors, which are essentially labile and variable, and in spite of the numerous influences which in an unbalanced physical system would cause inco-ordinate variation of pressure, the blood pressure under various conditions remains strikingly constant. It may be asked: What are the mechanisms which not only prevent such fluctuations, but also maintain and adjust the blood pressure in this precise and sensitive manner and so control and regulate the circulation and nutrition of the cells, tissues and organs of the body?

In the course of recent years, experimental investigations have demonstrated that the regulation of blood pressure is fundamentally an automatic, proprioceptive, reflex mechanism. In fact, the endovascular pressure itself regulates and adapts automatically the cardiac output, the circulating blood volume and the peripheral vascular resistance so well that the arterial pressure is maintained within normal limits. This automatic and proprioceptive regulation is effected mainly by the intermediation of the pressoreceptor innervation of different arterial and venous vascular areas.

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CARDIOAORTIC PRESSORECEPTIVE AREAS

The early experiments of Cyon and Ludwig,[1] as well as the more recent researches of Anrep and Starling,[2] of Ladon and myself,[3] and of Daly and Verney,[5] have shown that the cardioaortic vascular zone, especially the aortic arch, is provided

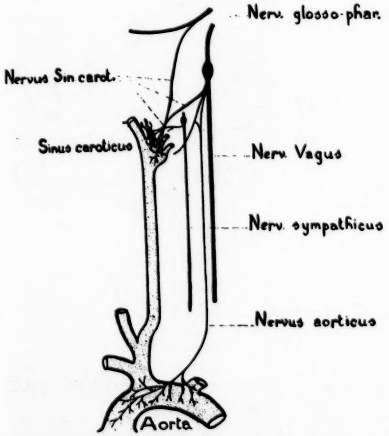

Figure 1. *Diagrammatic representation of the aortic and carotid-sinus nerves.*

with a pressoreceptive reflexogenic innervation (Fig. 1). Thus, when the endovascular pressure rises in the left heart and aortic arch, impulses ascend along the cardioaortic nerves, inducing reflex cardiac slowing, diminution of cardiac output and peripheral vasodilatation. On the other hand, when this pressure falls, the same pressoreceptors are no longer stimulated, and hence inverse vasomotor and cardiac reactions take place.

characterized by vasoconstriction and cardiac acceleration. These reflexes tend to keep a balance between hypertension and hypotension and to maintain normal arterial pressure.

CAROTID-SINUS PRESSORECEPTIVE AREA

One knows from the earlier reports of Cooper,[6] Marey,[7] Francois-Franck[8] and Hédon[9] that variations of carotid-cephalic arterial pressure produce immediate cardiac and vasomotor reactions. Carotid-cephalic hypotension, by clamping the common carotid arteries, for example, induces acceleration of the heart and somatic vasoconstriction. The opposite phenomena, namely cardiac slowing and somatic vasodilatation, are responses to carotid-cephalic hypertension. This regulation of blood pressure has been attributed in the past to the direct central influence of blood pressure acting on the cardioregulatory and vasomotor centers.

The work of Czermack[10] and Concato[11] and the observations of Sollmann and Brown[12] showed, on

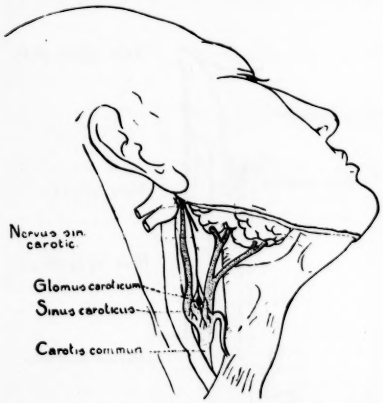

Figure 2. *Diagrammatic representation of the carotid sinus in man.*

the other hand, that the common carotid artery, particularly its bifurcation, is the origin of cardiovascular reflexes elicited by mechanical manipulations such as compression of the neck in the region of the carotid artery.

The experiments of Pagano[13] and Siciliano,[14] in 1900, had seriously damaged the doctrine of direct central regulation of blood pressure. But their experiments, which were incomplete and later contradicted by Kaufmann,[15] passed almost unnoticed, and the classic theory of the direct central regulation of blood pressure was still accepted until new investigations, performed mainly in the laboratory of Hering[16, 17] and in our laboratory,[3] clarified the forgotten observations of Pagano and Siciliano, and revealed the existence of the carotid-sinus mechanism by which the blood pressure is reflexly controlled.

The term carotid-sinus zone signifies a specially innervated area of the bifurcation of the common carotid artery (Fig. 1). Within this area in man (Fig. 2), dog, cat and rabbit are the origins of the internal and external carotid and the occipital arteries, the carotid bulb, which is a bulbous dilatation at the root of the internal carotid, and the carotid body or carotid ganglion, which lies be-

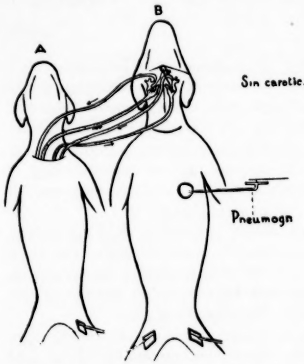

Figure 3. *Diagrammatic representation of the technic used for the profusion of the carotid sinus of Dog B by means of the carotid-jugular circulation of Dog A. The innervation of the carotid sinuses of Dog B is intact.*

tween the bifurcating arteries. This region is connected with the central nervous system by means of a distinct group of nerve fibers constituting the carotid-sinus nerves. These fibers originate in the adventitial coat of the arteries of the carotid bifurcation, mainly in the carotid bulb, and in the endothelial lining of the vessels of the carotid ganglion.

We may now consider briefly the functions of the carotid sinus in cardiovascular regulation.

With a technic (Fig. 3) developed in our laboratory,[3] one or both carotid sinuses of a dog are isolated in situ from the circulation, care being taken not to injure their innervation. These circulatory-isolated but innervated carotid sinuses are perfused with blood either by means of another dog, or with a Dale-Schuster pump. If the pressure inside the circulatory-isolated but innervated carotid sinuses is increased, we observe that this rise of intracarotid-sinus pressure is associated with a fall of the blood pressure in the general circulation (Dog B, Fig. 3). With the fall of arterial

pressure there is a slowing of the heart. Conversely, if the pressure is lowered in the isolated carotid sinus, the general blood pressure rises and the heart accelerates.

These experimental observations, in addition to many others, demonstrate that the general arterial blood pressure is automatically controlled by means of the endovascular pressure itself, acting on the pressosensitive nerve endings of the carotid sinuses.

The pressosensitivity of the carotid sinus is very great; it has indeed been shown by Koch[17] and ourselves[3] that in the carotid sinus of the dog, a deviation in either direction of even 1 mm. of mercury from the normal arterial pressure is sufficient to produce reflex compensatory changes in the general blood pressure. Moreover, Bronk and Stella[18] and ourselves[19] were able to register action-potential variations from the carotid sinus pressoreceptors at each systolic-diastolic variation of carotid sinus pressure.

Another group of experiments has permitted us[3] to show that the cardiovascular centers are not directly sensitive to variations in blood pressure. The reactions of the cardiovascular centers to changes in cephalic blood pressure are, indeed, entirely of carotid-sinus reflex origin. Only extreme anemia has a direct stimulating influence on the cardiovascular centers.

Let us analyze the components of these pressoreceptive mechanisms, namely the aortic and especially the carotid-sinus reflexes, which are responsible for homeostasis of the circulation.

The regulation of the heart frequency depends mainly on changes in the vagus tone and to a slight degree on changes in the sympathetic activity.

Control of the circulating blood volume and the peripheral vascular resistance is exercised by changes in the arterial, venous and capillary tone, and also by changes in the caliber of the arteriovenous anastomoses.

An increase in the carotid-sinus pressure produces reflex arterial, venous and capillary dilatation and a reduction in the caliber of the arteriovenous anastomoses, and a decrease in the cardiac output. A lowered carotid-sinus pressure produces, on the other hand, a reflex vascular constriction, an opening of the arteriovenous anastomoses, an increase in the circulating blood volume and thus an increase in the cardiac output.

By means of various experimental methods we were able to show[3] that these neurovascular reactions occur to a certain extent in the peripheral vessels, but mostly in the splanchnic area, that is to say the mesenteric vessels, spleen, liver and intestines.

Concerning the reflex regulation of the venous vascular tone by the aortic and carotid-sinus reflexes, the investigations of Fleisch,[20] and ourselves with Bouckaert and Dautrebande,[3] showed that the mesenteric veins, isolated from the general circulation but perfused in situ by means of Donegan's technic, contract reflexly during lowered aortic or carotid-sinus pressure and dilate if the sinus or aortic pressure is increased. Thus both the tone as well as the capacity of the abdominal venous reservoirs are reflexly and automatically controlled by the arterial blood pressure's acting on the pressosensitive carotid-sinus and aortic nerve endings.

The arterial and venous vascular tones are regulated, not only reflexly by means of neurovascular influences, but also by reflex alterations in the adrenine output. Our experiments[3] showed indeed that the arterial blood pressure, by acting on the pressoreceptors of the carotid sinus, regulates reflexly the secretion and discharge of adrenine, and thus controls further, in a humoral way, the vascular tone, the heart rate, the circulatory blood volume and probably also the caliber of the arteriovenous anastomoses. But we wish to point out that this suprarenal hormonal regulation of the circulation by the carotid sinus is only an accessory regulating mechanism. Indeed, the experimental observations of Nowak[21] and of ourselves[22] have shown that the reflex neurovascular regulation of the blood pressure occurs in the absence of the suprarenal medullary secretion. The increased discharge of adrenine in response to lowered carotid-sinus pressure is thus comparable to that which takes place in response to emergencies, as demonstrated by Cannon.[23]

Finally, another group of experiments with Bouckaert, von Euler and Dautrebande[3] has permitted us to assert that the carotid-sinus zone is provided with a reflexogenic innervation which is not only pressosensitive but also chemosensitive, so that changes in oxygen or carbon-dioxide content of the blood, or the presence of several pharmacologic substances in the arterial blood, can influence, by means of carotid-sinus reflexes, the activity of the cardiovascular centers and thus modify the general blood pressure. These chemoreceptors, which also regulate and influence reflexly the respiratory center, as first shown by our experiments, are localized in the glomus carcoticum, annexed to the carotid bifurcation. They are quite different from the pressoreceptors (Fig. 4). Figure 5 shows diagrammatically the general mechanisms concerned in the homeostasis of the blood pressure by means of the cardioaortic and carotid-sinus reflexes.

VENOAURICULAR, PULMOARTERIAL AND THORACICO-SPLANCHNIC PRESSORECEPTIVE AREAS

Another question presents itself at this point: Are the cardioaortic and carotid-sinus vascular zones the only regions provided with reflexogenic pressosensitivity and able to take part in the proprioceptive regulation of vascular tone and blood-pressure?

Experimental observations by Bainbridge[24] and McDowall[25] and the anatomical researches of

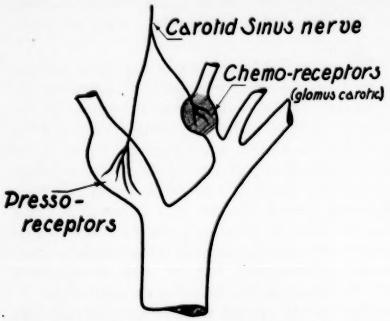

Figure 4. *Diagrammatic representation of the presso-chemoreceptors in the carotid sinus.*

Nonidez[26] have shown that the right auricle, as well as the venae cavae and pulmonary veins at their entrance into the heart, are provided with receptors through which pressure changes in the reflex bradycardia and arterial vasodilatation, while lowering of pulmonary arterial pressure provokes the opposite cardiovascular reactions.

The afferent pathways from the venoauricular and pulmonary arterial pressoreceptors are found in the cervical vagus nerves.

In 1929, we[3] observed that experimental suppression of the cardioaortic, carotid-sinus and cervical vagus nerves did not abolish completely the pressoreceptive mechanism of cardiovascular regulation. Indeed, in a dog deprived of the pressosensitive reflexogenic innervation of the cardioaortic, carotid-sinus, venoauricular and pulmonary zones, the elevation of general arterial pressure still induced compensatory reactions characterized by vascular dilatation and diminution of adrenine output. In the same animal, arterial hypotension started the opposite compensatory reactions of vascular constriction and increased adrenine secretion. This proprioceptive adaptation and control of vasomotor tone by the blood pressure is not of direct central encephalobulbar origin. The reactions still persist in the "spinal" animal, that is, the animal deprived of its encephalobulbar centers.

Experiments employing several different methods have permitted us, with the collaboration of Bouckaert, Farber, Hsu and Wierzuchowski,[28, 29] to show that in the dog deprived of carotid-sinus, cardioaortic, pulmonary and venous pressosensitive zones, as in the spinal animal, the proprioceptive regulation of vascular tone still occurs, through pressosensitive reflexes originating mostly from the vascular territory of the celiac and mesenteric arteries, and secondarily from the territory of the

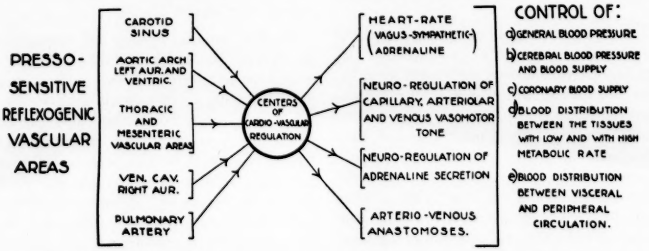
PRESSO-SENSITIVE REFLEXOGENIC VASCULAR AREAS
CAROTID SINUS
AORTIC ARCH LEFT AUR. AND VENTRIC.
THORACIC AND MESENTERIC VASCULAR AREAS
VEN. CAV. RIGHT AUR.
PULMONARY ARTERY
CENTERS OF CARDIO-VASCULAR REGULATION
HEART-RATE (VAGUS-SYMPATHETIC-ADRENALINE)
NEURO-REGULATION OF CAPILLARY, ARTERIOLAR AND VENOUS VASOMOTOR TONE
NEURO-REGULATION OF ADRENALINE SECRETION
ARTERIO-VENOUS ANASTOMOSES.
CONTROL OF:
a) GENERAL BLOOD PRESSURE
b) CEREBRAL BLOOD PRESSURE AND BLOOD SUPPLY
c) CORONARY BLOOD SUPPLY
d) BLOOD DISTRIBUTION BETWEEN THE TISSUES WITH LOW AND WITH HIGH METABOLIC RATE
e) BLOOD DISTRIBUTION BETWEEN VISCERAL AND PERIPHERAL CIRCULATION.

Figure 5. *Schematic representation of the actions of the pressosensitive reflexogenic vascular areas on the general mechanisms concerned in the homeostasis of blood pressure.*

venous system may influence and regulate reflexly the heart rate and the arterial vascular tone.

More recently Schwiegk[27] has shown that the branches of the pulmonary artery are also endowed with reflexogenic pressosensitivity regulating the heart rate and arterial vasomotor tone. Increase of pressure in the pulmonary arterial system causes thoracic arteries. These spinal vasomotor reflex responses to variations in blood pressure may arise in part from the mesenteric pressoreceptors, discovered in the pacinian mesenteric corpuscles by Gammon and Bronk,[30] who have recently registered afferent action currents related to variations in blood pressure from these structures.

These experiments raise another final question, Are all vessels provided with reflexogenic pressosensitivity? There are experimental facts which make it possible to answer in the negative. Proprioceptive regulation of general vascular tone is effected through reflexes, the pressoreceptors for which are situated only in certain well-localized vascular areas, namely the carotid sinus and the cardioaortic, venoauricular, pulmoarterial and thoracicosplanchnic zones. In this connection it may be worth mentioning that a decrease of blood pressure in other vascular areas may, however, induce only local or regional vasomotor reactions. Indeed, it has been shown by Bayliss[31] and others that clamping of an artery provokes a vasodilatation in the tissues which it normally supplies. These vascular reactions, however, are not involved in the pressoreceptive regulation of the general blood pressure but only in the local "nutritive" regulation of the blood supply.

ROLE OF THE PRESSORECEPTIVE MECHANISMS OF CARDIOVASCULAR REGULATION IN THE MECHANISM OF CIRCULATORY COLLAPSE

Let us now consider, in the light of the pressoreceptive mechanisms governing and controlling blood pressure, the reactions of the organism against disturbances in blood pressure.

The first fact to be borne in mind is that the normal organism has at its disposal a certain reserve of blood pressure. This reserve is liberated and used progressively, when depression of the circulation occurs, to maintain or to readjust the general blood pressure at its normal level. This liberation takes place by means of the aortic and carotid-sinus pressoreceptive mechanisms, which are absolutely necessary for the restoration of pressure when the arterial tension is disturbed to a pathologic degree. This fact was clearly demonstrated by the experiments of Koch[17] and our experiments with Bouckaert and Bert.[3] Removal of no more than 10 per cent of the total blood volume of a normal dog provokes a transient fall of blood pressure, which returns to normal within a few minutes. The normal organism is thus able to compensate readily for the arterial hypotension provoked by hemorrhage. But if the aortic and carotid-sinus pressoreceptive mechanisms are excluded, by central or peripheral paralysis or depression, loss of 10 per cent of the blood volume produces a profound and lasting fall of blood pressure, a state of circulatory collapse. We may conclude from these experimental observations that any cause or factor which depresses or paralyzes the reflex proprioceptive regulation of the circulation by the aortic and especially the carotid-sinus reflexes will decrease or abolish the normal compensatory reactions of the organism against arterial hypotension. A number of such factors have been experimentally tested. Thus experiments in our laboratory[3] have shown that hypocalcemia, produced by citrate or oxalate injections, or by parathyroidectomy, depresses or paralyzes the carotid-sinus vasomotor reflexes. On the other hand, the restoration of the calcium content of the blood and tissues to normal, as by the administration of calcium salts, parathormone or vitamin D, will restore the normal carotid-sinus regulation of circulation.

Spinal anesthesia, deep general anesthesia with ether or chloroform, the injection of barbiturates, the administration of morphine and other narcotic drugs, the injection of histamine, ergotamine or several dioxane derivatives, will very often depress considerably, or even abolish, the pressoreceptive regulation of blood pressure and circulation. Various forms of trauma, pulmonary hyperventilation and severe anoxemia may have the same depressant or paralyzing effects. Experimental observations have shown that by trauma or during spinal anesthesia the aortic and carotid-sinus vasopressor reflexes may be absent although the general blood pressure is at a normal level. Under such conditions factors which ordinarily cause a slight fall of blood pressure may produce a break in the homeostasis of the circulation and thus a deep and permanent fall of blood pressure, a condition of circulatory collapse.

It is therefore possible to suggest that one of the main causes of circulatory collapse after trauma and during general or spinal anesthesia is related to the central or peripheral paralysis or acute depression of the pressoreceptive mechanisms concerned in homeostatic cardiovascular regulation.

To eliminate very important causes of circulatory collapse, it is advisable therefore to avoid anoxemia, high spinal anesthesia and the administration of narcotics. It is obviously impossible always to avoid these causes of depression or paralysis of the automatic reflex regulation of the circulation. Fortunately we know of certain chemicals and drugs which may stimulate, sensitize or restore the pressoreceptive circulatory reflexes. For example, oxygen, carbon dioxide, strychnine, adrenalin, ephedrin and physostigmine are, under some conditions, definite stimulants of the pressoreceptive reflexes regulating and maintaining the blood pressure.

ROLE OF THE PRESSORECEPTIVE MECHANISMS OF CARDIOVASCULAR REGULATION IN THE ADAPTATION OF BLOOD FLOW AND BLOOD SUPPLY

From a hydrodynamic point of view, the amount of blood irrigating any given part of the body depends mainly upon the tonus of the arteriocapillary vessels in that part and also upon the

blood pressure in its afferent arteries. The phenomenon is a simple physical one, but its causes, which are physiologic, lead to a more complex problem, namely the relations between the mechanisms regulating blood pressure and those adapting the peripheral circulation to the nutritional needs of tissues and organs.

It is known that when tissues or organs are in a condition of high activity, their nutritional and respiratory requirements are increased, while their vessels are dilated mainly through the local, direct and reflex influences of metabolites and temperature.

The experiments of Rein[32] and our observations with Bouckaert[33] have shown that the blood vessels of organs in such periods of "nutritional" vasodilatation are temporarily "immune" to any vasoconstrictor influences, nervous or adrenic, which may be set up by the pressoreceptive reflexes concerned with blood-pressure homeostasis. Thus the vasoconstrictor impulses regulating blood pressure have very little or no influence on vessels in a state of nutritional vasodilatation; in fact the principal and often the only constrictor effect is on the vessels of resting organs. By this mechanism, blood is shifted readily from regions where respiratory and nutritional needs are slight to tissues whose needs are greater, although the general and afferent blood pressures are maintained at normal levels or regulated at higher values. Thus it is seen that the pressoreceptive mechanisms of blood-pressure homeostasis assist also in the adaptation of the "nutritional" blood supply in tissues and organs.

Another group of experiments with Bouckaert[33] disclosed that the cerebral vessels do not participate actively in the pressoreceptive regulation of general blood pressure. In fact, the brain always conducts itself as an organ in a state of high activity, its blood supply depending essentially upon the afferent blood pressure and upon local and humoral chemical factors. The cerebral blood vessels thus always behave as vessels of an organ the nutritional needs of which are elevated, so that if necessary, other organs in a state of metabolic rest may sacrifice some of their blood to the cerebral circulation.

Indeed, in periods of arterial hypotension, pressosensitive reflexes, particularly from the carotid sinus, divert blood from the peripheral and splanchnic areas toward the cerebral circulation. During hypertension, on the other hand, some blood is diverted from the brain, by the same reflexes, toward the splanchnic and peripheral zones (Fig. 6). In these mechanisms the peripheral cephalic tissues and the thyroid gland play an important role. Indeed, Rein and his co-workers[34] have shown that an increase in the carotid-sinus pressure induces reflex thyroid vasodilatation, thus diverting a certain amount of carotid blood through the thyroid gland. Furthermore, our experiments[3, 33] demonstrated that the vessels of the extracranial tissues constrict reflexly when the carotid-sinus pressure is lowered; thus the extracranial blood is shifted toward the cerebral circulation through the internal carotid artery, and also through the large anastomotic arterial branch connecting the extracranial and the intracranial

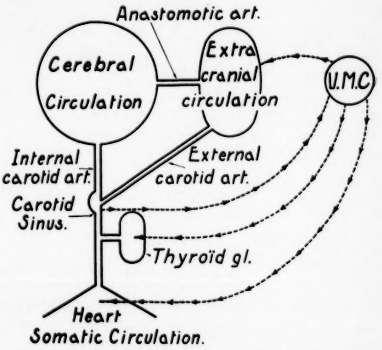

Figure 6. *Diagrammatic representation of the regulation of the cerebral circulation by means of carotid-sinus reflexes. The role of the extracranial and thyroid circulations in the regulation of cerebral pressure and blood supply is indicated. Attention is called to the anastomotic artery which connects the extracranial circulation with the cerebral circulation. V.M.C. = nerve centers of cardiovascular circulation.*

blood vessels. When the carotid-sinus pressure is increased, opposite reactions are set up. We have convincing evidence that the extracranial circulation plays a leading part in the regulation of the cerebral circulation, mainly in emergencies. The influences of the carotid-sinus reflexes on the cerebral circulation were studied recently by Ask-Upmark,[35] under the direction of Forbes, and by Bouckaert and Jourdan[36] in our laboratory. Researches by Weiss and his collaborators[37, 38] demonstrated furthermore the function of the carotid-sinus reflexes in the pathogenesis of cerebral syncope and certain convulsive states.

The proprioceptive mechanisms of blood-pressure regulation protect not only the cerebral circulation but also the heart against variations of arterial pressure and blood supply. In the heart, an organ always at work, the coronary blood supply depends in part upon the aortic pressure. The homeostasis of the aortic pressure and the pressure in the left

auricle and ventricle depend mainly upon the reflex pressosensitivity of the left ventricle itself, the aortic arch and the pulmonary artery. Again the pressosensitivity of the pulmonary artery, the venae cavae, the pulmonary veins and the right auricle regulates particularly the venous pressure, the pulmonary blood pressure and, thus, the pressure in the right auricle and ventricle. It appears, therefore, that the reflex pressosensitivity of the veins

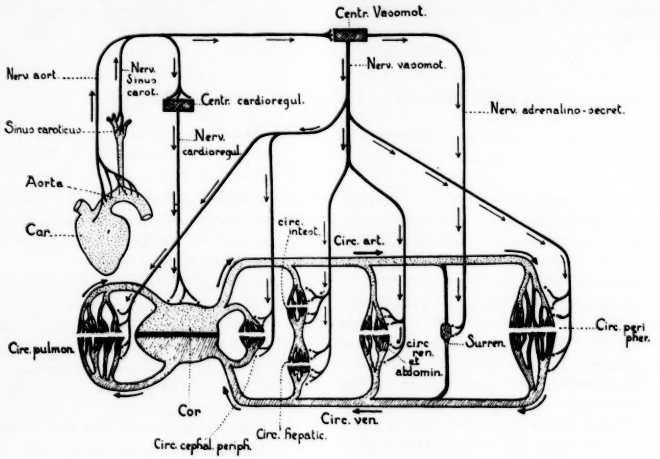

Figure 7. *Diagrammatic representation of the main physiologic functions of the pressosensitive reflexogenic vascular areas in the homeostasis of circulation.*

and the pulmonary artery plays perhaps a significant role in the etiology of some forms of pulmonary hyperemia, pulmonary edema and failure of the right heart.

The pressosensitive reflexes of the mesentericointestinal zones, we believe, act chiefly in the local, segmental and regional distribution of blood between the abdominal and peripheral circulation. Figure 7 shows diagrammatically the main physiologic functions of the pressosensitive reflexogenic vascular areas in the homeostasis of circulation.

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EXPERIMENTAL ARTERIAL HYPERTENSION

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IT IS known that the blood pressure is regulated reflexly by the pressoreceptive mechanisms, chiefly those of the cardioaortic and carotid-sinus vascular areas.[1–5]

The cardioaortic and carotid-sinus nerves are not only the means of the automatic pressoreceptive reflex regulation of general arterial pressure but also the *buffer nerves* or *moderator nerves* of the blood pressure. In fact, functional suppression of these nerves immediately produces an intense arterial hypertension. In dogs the pressure may rise from 130 mm. of mercury to 300 mm. as a consequence of the section of the nerves. This marked increase of blood pressure is caused by the release of vasoconstrictor and cardioaccelerator centers, the activity of which, under normal conditions, is permanently moderated reflexly by the cardioaortic and carotid-sinus nerves. The moderator tone of these nerves is maintained and regulated by means of the pressure and the chemical constitution of the blood acting on the pressosensitive and chemosensitive nerve endings of the corresponding vascular areas.[4]

In dogs we[4] have observed arterial hypertension maintained at 250 to 300 mm. of mercury for nine to twenty-six months after section of the cardioaortic and carotid-sinus moderator nerves. The same observation has been made by Nowak[7] on dogs up to three years after section of these nerves. If, in a certain number of animals, section of the cardioaortic and carotid-sinus nerves produces only a temporary hypertension, this is explained by the presence of accessory fibers of the cardioaortic nerves in the vagus or, as we believe, occurs because in certain animals the pulmonary and mesentericointestinal pressosensitive nerves may take over the moderator function of the cardioaortic and carotid-sinus nerves.

The degree of hypertension which develops after section of the moderator nerves depends mainly upon the sympathetic vasopressor tonus. This tonus is an index of the state of activity in the cortical psychomotor, hypothalamic and bulbar centers, as well as the result of a series of humoral and cellular factors amongst which are the blood supply to the sympathetic centers and the carbon-dioxide and oxygen content of the blood.

With Bacq, Brouha and Bouckaert we[8] have indeed shown the role of the vasopressor sympathetics in the mechanism of experimental hypertension produced by exclusion of the moderator nerves. In dogs, excision of the sympathetic paravertebral ganglionic chains, from the stellate ganglia down to the pelvic ganglia, performed in three operative stages, prevents or causes the disappearance of this type of experimental hypertension. We noticed also that the pressoreceptive reflexes of cardioaortic and carotid-sinus origin, regulating vascular tone and arterial pressure, are absent in the totally sympathectomized dog. No vasomotor response could be detected either by reflex or direct stimulation of the vasomotor centers in dogs up to several months and as long as one year after total removal of the sympathetic chains. The general arterial pressure, the thermoregulation and the psychic and motor behavior of these totally sympathectomized dogs were, however, normal, as was also shown by Samaan[9] and Brouha, Cannon and Dill.[10] After each operative stage of the sympathectomy, the animal passes, of course, through a period of one or two weeks of general depression;

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the blood pressure is lowered as a consequence of the removal of an important part of the vasomotor tone of central origin. Soon, a progressive rise in peripheral vascular resistance is restored by means of a peripheral vasopressor mechanism, originating in part in the sympathetic synapses located more peripherally to the paravertebral chains, and in the vascular wall. Thus, the general blood pressure returns to normal levels. At complete rest, the blood pressure of the totally sympathectomized dog may, however, be somewhat under the mean level of normal dogs as a consequence of the slow rate of the heart deprived of its sympathetic innervation and controlled only by the vagal cardioinhibitory tone.

The question arises: Is the experimental hypertension induced by exclusion of the moderator nerves related to permanent clinical hypertension? We can answer at least a part of this question in the negative. This form of experimental hypertension varies indeed from the typical nephropathic variety, but resembles more closely the so-called "essential neurogenic hypertension." This suggests that a functional deficiency of the moderator nerves, either in the region of the vasoconstrictor centers or in that of the peripheral pressoreceptors or in that of the peripheral vasoconstrictor nerves, is the underlying cause for essential hypertension, and that surgical interruption of the efferent vasopressor sympathetic nerve paths is indicated in this condition of "neurogenic" hypertension.

Recently, indeed, Allen and Adson[11] concluded from their clinical observations "that the bilateral resection of the splanchnic nerves, celiac ganglion and two upper lumbar sympathetic ganglia, for essential hypertension, can be carried out without undue risk; that patients are not disabled as a result of operation; and that the blood pressure of many of them is significantly reduced by operation. Other patients note amelioration or relief of symptoms even when their blood pressures have not been significantly reduced."

A second method which produces experimental hypertension was described by Dixon and Heller.[12] These authors injected a kaolin suspension into the cerebral ventricles or into the subarachnoid space. The mechanical compression thus produced led to cerebral anoxemia, which particularly sensitizes the vasopressor centers to the stimulating action of carbon dioxide. That anemia and anoxemia may stimulate these centers has been demonstrated by several experimenters. Raab[13] has shown that central anemia, especially central acidosis, stimulates the vasopressor centers and increases their sensitivity to the humoral carbon dioxide. In our laboratory Nowak and Samaan,[14] by perfusing the isolated head connected to the trunk only by the spinal vasomotor pathways, also produced evidence that acute anemia alone in the cerebral circulation provokes an intense somatic arterial hypertension. Experiments of Nowak[15] further showed that the permanent decrease of cerebral blood supply by ligature of the carotid vertebral and spinal arteries may induce in dogs a condition of permanent arterial hypertension. These observations support the views of Kylin[16] and Raab,[17] who believe that some forms of clinical hypertension are related to an anoxic process, such as endarteritis, localized in the region of the vasopressor centers.

Recently Goldblatt and his co-workers[18] made a very important observation. They showed that in the dog and the monkey renal ischemia through permanent, incomplete compression of the renal arteries caused chronic, progressive arterial hypertension, with elevation of both the systolic and the diastolic pressures. This fact has largely been confirmed by Page[19] and others, and in our laboratory by Elaut,[20] Bouckaert and ourselves.[21] In our experiments we obtained permanent hypertension in dogs reaching 245 mm. of mercury. Furthermore, we observed that the carotid-sinus vasopressor and hypertensive reflexes were especially active in such dogs. Page[19] and Collins[22] observed that, in dogs, excision of the extrinsic renal nerves alone does not prevent experimental hypertension due to renal ischemia. The experiments of Goldblatt and his collaborators[23] showed that neither excision of one adrenal, with denervation and destruction of the medulla of the other, nor excision of the splanchnic nerves and the lower dorsal ganglia, which constitute an important part of the sympathetic vasopressor system, prevents or cures experimental hypertension produced by renal ischemia.

More recently our experiments performed with Bayless, Bouckaert, Elaut and Samaan[24] showed that complete exclusion of the whole sympathetic ganglionic chains in dogs also fails to prevent the arterial hypertension following renal ischemia. The same observations have been made by Freeman and Page[25] and Alpert, Alving and Grimson.[26]

Our experiments suggest that the arterial hypertension induced by renal ischemia may be due to a humoral factor which increases the excitability of the peripheral blood vessels to constrictor stimulations, mainly to the neurogenic vasoconstrictor influences. The same humoral factor induces, on the other hand, a direct peripheral vasoconstriction and a disturbance in the physiologic mechanisms of the pressoreceptive reflex regulation of blood pressure. Sympathectomy up to the total removal of both ganglionic chains neither prevents nor cures this experimental nephrogenic hypertension. But, as pointed out by Goldblatt, although the results of these investigations are in

agreement with the conclusions of Prinzmetal and Wilson[27] and of Pickering[28] concerning the part played by the vasomotor system in human hypertension of nephropathic origin, they do not necessarily exclude some beneficial effects of operation on the vasomotor system or on the suprarenal glands in some cases of human hypertension. Further experimental and clinical studies are necessary to evaluate adequately the indications and the results of surgical intervention.

A fourth method of producing experimental arterial hypertension is that indicated by Appelrot.[29] Further researches along this line were carried out by Handovsky in our laboratory, with the collaboration of Goormaghtigh[30] of the Department of Pathology. Their studies show that the repeated administration of relatively large doses of vitamin D_2 (calciferol) in dogs provoked a progressive augmentation of the arterial blood pressure. It was noticed also that the dogs were very sensitive to the hypertensive action of adrenalin. In the kidneys and elsewhere, certain vascular lesions, mainly arteriolonecrosis, developed which could be almost superimposed upon those found in human eclampsia, diphtheria and scarlet fever. It is furthermore interesting that vitamin D_2, which provokes arteriosclerosis in the rat and rabbit, does not induce the same vascular sclerotic changes in the dog, unless one deprives this animal of its thyroid gland. Thus, in the thyroidectomized dog the administration of vitamin D_2 provokes arterial hypertension, and with it intense sclerosis of the aorta and its major branches and typical lesions in the smaller arteries. These observations are interesting, since this form of experimental hypertension, characterized by high pressure and vascular lesions and sclerosis, resembles still more closely some types of human hypertension. As a matter of fact, many pathologists and clinicians have referred to a relation between the thyroid gland, arteriosclerosis and hypertension. The researches of Aub and his collaborators[31] showed especially the close relation between the thyroid and calcium metabolism.

These different groups of researches show once more the bonds closely uniting experimental physiology, pathology and surgery in attempts to elucidate the mechanisms regulating arterial blood pressure and the causes of hypertension. The knowledge thus gained, it is hoped, will lead to a more adequate treatment of this very important disease.

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ROLE OF THE CARDIOAORTIC AND CAROTID-SINUS NERVES IN THE REFLEX CONTROL OF THE RESPIRATORY CENTER

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CIRCULATION and respiration are two very closely interdependent physiological functions. We shall discuss some of the functional relations and physiological co-ordinations between them.

Experiments performed with J. F. Heymans[1] have shown that changes in intravascular pressure acting on the pressosensitive nerve endings of the cardioaortic area regulate reflexly not only the activity of the cardiovascular centers but also that of the respiratory center. It has been shown by these experiments that an increase of blood pressure in the cardioaortic area induces a reflex inhibition of the respiratory center, or even apnea, while lowering of the cardioaortic pressure provokes a reflex stimulation and hyperpnea.

It is known from the observations of Cooper[2] and others that a decrease in the cephalic blood pressure and blood flow, by occluding the common carotid arteries, produces hyperpnea, while an increase inhibits the activity of the respiratory center. These facts were taken by several physiologists as evidence that the activity of the center is influenced and regulated directly by changes in its blood supply. As early as 1900, however, Pagano[3] pointed out that respiratory stimulation is much more marked after the occlusion of the carotids than after that of the vertebral arteries, although the latter are more important for the central blood supply. He ascribed these changes to respiratory reflexes of carotid origin.

The investigations of Hering,[4] Danielopolu,[5] Koch[6] and ourselves[7] have shown that electrical or mechanical stimulation of the carotid sinus provokes reflex excitation, sometimes inhibition, of the respiratory center.

The findings of Moissejeff[8] and Koch[6] and the results obtained in our laboratory[7] have further demonstrated that blood-pressure changes in the pressosensitive area of the carotid sinus, just as in the aortic arch,[1] affect reflexly the activity of the respiratory center: a rise of pressure producing reflex respiratory inhibition, even apnea, while a lowering provokes reflex hyperpnea. A number of our observations[7] involving several different experimental methods show that the immediate changes in the activity of the respiratory center which occur after modifications of blood pressure and blood flow in the cephalic arterial circulation are due to reflexes arising in the carotid sinus, and not to changes in the central blood supply. The latter directly affect the activity of the respiratory center only when extreme pathologic changes take place. Thus acute anemia may stimulate the respiratory center directly, while an increase in central blood flow occurring after a period of anemia or anoxemia may directly inhibit it.

Experiments performed with Bouckaert, Farber, Nowak and Jourdan[9] showed further that the respiratory center is very resistant to central anemia or anoxemia. Its activity can, indeed, be revived, as demonstrated by these experiments, even after a complete arrest of the circulation for thirty minutes.

Not only the arterial pressure but also the venous pressure may influence the respiratory center reflexly. Harrison, Harrison and Marsh[10] have shown that an increase of venous pressure in the vena cava and the right auricle provokes a reflex increase in respiratory activity, even a hyperpnea. The reflex stimulation of the respiratory center by lowered aortic and carotid-sinus arterial pressure and increased vena cava pressure may perhaps give an explanation of the mechanisms and origins of some forms of pathologic respiratory modifications; cardiac dyspnea may perhaps be related to these respiratory reflexes.

Not only the pressure changes but also the chemical changes of the blood in contact with the cardioaortic and carotid-sinus regions act reflexly on the activity of the respiratory center.

In a first group of experiments, performed in 1926, we[1] could demonstrate that asphyxia of only the somatic or cardioaortic circulation provokes reflex stimulation of the respiratory center by the intermediation of the cardioaortic nerves.

Later, these experiments were extended, with the collaboration of Bouckaert and of Dautrebande,[7] to the vasosensory zones of the carotid sinus. If the isolated carotid sinuses, with their innervation intact, are perfused by means of either a Dale-Schuster pump or a second perfusing dog, it may be shown that an increase of carbon dioxide in the perfusing blood produces reflex stimulation of the respiratory center. Poorly oxygenated or anoxic blood acting on the carotid sinus also brings about this result. Dogs, cats or rabbits with aortic and carotid-sinus nerves suppressed show little or

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no hyperpnea during acute anoxemia. Thus, lack of oxygen stimulates the respiratory center mainly reflexly through the carotid-sinus chemosensitivity, and not by a direct central action.

A consideration of the influence of carbon dioxide reveals that, although the respiratory reflexes of carotid-sinus origin may be elicited by alterations of carbon-dioxide tension in the blood within the physiological range, it acts on the respiratory center not only through the carotid-sinus reflexes but also by direct central action.[7] In other words, after interruption of the aortic and carotid-sinus reflex paths, the carbon-dioxide content of the blood still affects and controls respiration. Experiments of Wright[11] showed, however, that rabbits deprived of their carotid-sinus mechanisms are much less sensitive to carbon dioxide as a respiratory stimulant. Samaan and Stella[12] registered the action currents of the carotid-sinus chemoreceptors exposed to different humoral concentrations of carbon dioxide, and observed very striking differences in the activity of these reflexogenic chemoreceptors, although the changing carbon-dioxide content of the blood was kept within physiological limits.

From recent experiments Comroe and Schmidt[13] conclude that the hyperpnea of carbon dioxide and acid excess is more central than reflex, while anoxemic hyperpnea is mainly or entirely reflex. We believe that further confirmatory evidence should be adduced before this opinion can be fully accepted.

The chemoreceptors of the carotid sinus are sensitive not only to physiologic chemical stimulants, but also, as first shown by our experiments with Bouckaert and Dautrebande,[7] to pharmacologic substances. Cyanides, nicotine, lobeline, sulfides, acetylcholine, and so forth, act on these receptors producing a marked reflex hyperpnea. Small doses of these substances may provoke respiratory reflexes, by acting on the chemoreceptors of carotid-sinus nerve endings, while the same doses have no direct action on the respiratory center. This fact can be shown best by the following experimental method. One carotid bifurcation is denervated and the efferent blood vessels, except the internal carotid, are ligated in order to shunt the blood to the central nervous system. The nerves of the opposite carotid bifurcation remain intact, but the internal and external carotid and the occipital arteries are ligated, in order to limit the circulation through the carotid body by way of the small afferent branches arising mainly from the occipital artery. With this preparation one observes that the respiratory center is reflexly stimulated by the injection into the common carotid, with the normally innervated bifurcation, of solutions containing the above-mentioned drugs, whereas the same solutions are without effect when injected into the denervated carotid artery which carries blood to the respiratory center. There is convincing evidence that the chemoreceptor nerve endings of the carotid bifurcation are much more sensitive to certain chemical substances than the respiratory center itself, which is influenced directly only by much higher doses. This specific sensitivity was shown by several of our experiments to be localized in the carotid body, in contrast to the pressosensitive nerve endings, which are situated mainly in the wall of the dilated portion of the internal carotid artery.

It has further been shown that the chemoreceptors of the carotid body affect and reflexly control not only the respiratory center but also the cardiovascular centers.[7]

Our observations on the role and functions of the chemoreceptors of the carotid body in the reflex control of respiration, whether by pharmacologic or physiologic agents, have been largely confirmed by numerous investigators, amongst them Wright and his co-workers,[13, 14] Samaan and Stella,[12] von Euler and Liljestrand,[15] Schmidt,[13, 14] Gollwitzer-Meier,[17] Gemmill and Reeves,[18] Gesell and his co-workers,[19] Gayet, Bennati and Quivy[20] and many others. These experimental facts demonstrate a new and, to us, an important mechanism of physiologic and pharmacologic reflex control of the respiratory center.

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REGIONAL ENTERITIS

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THE purpose of this paper is to summarize briefly the more important literature of regional enteritis and to add two typical cases.

Interest in and recognition of regional enteritis has been general only since its elucidation in 1932 by Crohn, Ginzburg and Oppenheimer;[10] but the disease is not new. Combe and Saunders[8] in 1813 described the typical gross lesions of the process, and in 1828 Abercrombie[1] reported symptoms and postmortem findings of another indubitable case. Attention was directed by Braun[5] in 1908 to the nonspecific intestinal granulomas, most of which had theretofore been called hyperplastic tuberculosis. Later, Tietze,[43] Moschcowitz and Wilensky,[35] Körte,[31] Mock,[34] and many others wrote about these granulomas, but only in 1932 was regional enteritis clearly differentiated from the larger and more general group by Crohn and his co-workers.

The original description is of "a disease . . . of the terminal ileum" which was named "regional ileitis." Other names suggested have been chronic cicatrizing enteritis (Harris, Bell and Brunn[25]), chronic ulcerative enteritis (Corr and Boeck[9]), ileocolitis (Erb and Farmer[14]), ileocecal pseudotumor (Gagliardi[19]), ileocolitis ulcerosa chronica (Rosenblate et al.[40]) and, simpler and perhaps better, regional enteritis (Brown, Bargen and Weber[6]). All these names refer to a disease predominantly of young adult, Semitic males, although apparently no person is immune.

The symptomatology of the disease is very variable and correlates logically with the pathologic lesions, so that these aspects may best be considered together. Crohn et al.[10, 11, 12] and all subsequent authors have stressed the natural division of the manifestations of regional enteritis into those of acute peritoneal irritation, ulcerative enteritis, intestinal obstruction and fecal fistula. By no means do all these phases appear in each case; nor need this be the order of their succession, although it is the usual one.

The initial phase of peritoneal irritation, often indistinguishable from that due to acute appendicitis, may be entirely lacking. Laparotomy at this stage reveals one or more sections of ileum, usually terminal, — or, much less frequently, of jejunum or colon, — which are thickened, edematous and covered with reddened peritoneum. There is lymphadenitis, and usually lymphedema, of the regional mesentery; there is usually a little clear peritoneal transudate. The whole picture justifies the clinical findings: pain and tenderness, usually in the right lower quadrant; moderate fever, usually below 102°F.; and slight leukocytosis, usually between 10,000 and 15,000.

The enteritis phase may be severe and predominant over all others, or it may be lacking. Pathologically, the lesion at this stage shows progression, with even more peritoneal reaction, possibly engendering a fibrinoplastic transudate and increased edema of the mesentery and of the intestinal wall, with further thickening of the latter and corresponding reduction of the lumen. The thickened mucosa may show areas of papillary or polypoid hyperplasia, but characteristically presents small, linear ulcerations along the mesenteric border, which tend slowly to perforate into the mesentery with formation of more or less extensive, intramesenteric abscesses, single or multiple. Clinical findings resemble those of ulcerative colitis. There is usually diarrhea of from two to six poorly formed stools daily, examination of which may re-

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veal the presence of blood, pus and mucus. Leukocytosis is usually definite, and fever may vary from 99° to 103°F. or more, typically with frequent and prolonged remissions. Diffusion of tenderness and of pain, often cramp-like, appears along with weight loss, secondary anemia and asthenia.

Obstruction is seldom more than moderate, and often is absent entirely, although it may become complete, necessitating enterostomy. It results from the progressive thickening of all the intestinal layers, and elicits the symptoms of mechanical obstruction, incomplete or complete, from any other cause; that is, crampy pains, nausea and vomiting, varying abdominal distention with borborygmi or perhaps visible intestinal patterns, and usually constipation or obstipation. With subacute obstruction, however, constipation may alternate with periods of profuse diarrhea. A mass may be felt, usually in the right lower quadrant, and it is often sausage-shaped.

Fistula formation may precede any sign of obstruction, may follow it (usually when the process is of long standing), or may never supervene at all. It usually results from drainage of a mesenteric abscess into an adjacent bowel loop, most often thus uniting the terminal ileum and sigmoid. Perforation of the ulcers is always too slow to produce diffuse peritonitis. Although internal fistulas are more common, external fistulas with persistent fecal discharge may form as the result of direct rupture through the abdominal wall or, more usually, after appendectomy or drainage of an appendiceal abscess at the time of which the true condition was not recognized nor resection performed. At this stage, a mass can always be palpated.

Microscopic examination of the lesions is more interesting than helpful. It shows merely an inflammatory hyperplasia of all layers from mucosa to subserosa and peritoneum, each being diffusely infiltrated by chronic, nonspecific granulation tissue containing fibroblasts and endothelial histiocytes. Ulcerations are lined by the same type of tissue, infiltrated with lymphocytes and polymorphonuclear leukocytes. There are none of the lesions typical of any of the specific granulomas, and only enough foreign-body giant cells to account for the persistent suggestion of the tubercle bacillus as the etiologic agent.

Crohn et al.[10] in 1932 said that the etiology of the disease was unknown, and this statement still holds, although many suggestions as to the etiology have been proffered. Clute,[7] Bell,[3] Holman[27] and others have suggested primary infection of mesenteric nodes or lymphatics, with secondary spread to the intestinal wall. Holman bases his hypothesis on an early case of regional enteritis in a six-year-old child, in which there was no mucosal ulceration whatever but extensive, diffuse edema of the mesentery and lymph nodes. The relation of appendicitis to the lesion has been discussed by many writers, as has that of the appendicitis fibroplastica of Laewen (Fischer and Lürmann;[18] Ravdin and Rhoads[38]) because of its histologic similarity; but the consensus is that they are not of significance. Specific infective agents have been suspected, such as *Bacillus W,* a variant of *B. coli,* by Erb and Farmer;[14] an anaerobic streptococcus, by Mixter;[33] and *B. dysenteriae,* by Felsen and Gorenberg.[16] Tubercle bacilli have never been demonstrated in the lesions, nor has guinea-pig inoculation from the lesions ever been productive of tuberculosis. Mechanical factors, including interference with the blood supply by recurrent volvulus or self-reducing intussusception (Bell;[3] Bockus and Lee[4]), fecal stasis with thereby increased absorption of infective agents (Probstein and Gruenfeld[37]) and trauma (Gagliardi[19]) have been suggested. Gisbertz[22] believes impaired innervation of intestinal and mesenteric capillaries to be at fault, this leading to increased permeability, tissue edema and secondary infection. Recently Crohn[11] has described the coincidence of regional enteritis in a fourteen-year-old boy and his thirty-two-year-old sister, raising the question of the probability of congenital predisposition or of some transmissible etiologic factor, and calling to mind the theory of virus infection as advanced by Razzaboni, cited by Ravdin and Rhoads.[38] Particularly significant seems the work of Reichert and Mathes[39] on dogs in which, using a technic developed by Sabin, they injected sclerosing agents into the ileal lymphatics and obtained chronic lesions similar to those of regional enteritis in every respect except for the absence of ulcerations.

Diagnosis of regional enteritis can often be suspected if the disease is kept in mind, especially where there is a history of subacute onset of abdominal pain with symptoms of mild obstruction. Final diagnosis requires a barium meal with x-ray plates which should be taken serially every hour from the third to the ninth, thus making possible the demonstration of constant filling defects of the involved bowel with proximal distention, the "string sign" described by Kantor[30] (usually), and perhaps the granular, "cobblestone" appearance of the mucosal pattern which is pathognomonic of the disease. Before definite diagnosis by this means or by laparotomy, differentiation from ulcerative colitis, ileocecal or mesenteric tuberculosis, appendicitis, lymphosarcoma, Hodgkin's disease, actinomycosis and carcinoma must be considered.

The subject of treatment of regional enteritis has been much discussed. The most usual method

is primary resection of the lesion or, if the patient's condition requires a lesser procedure, lateral anastomosis with short-circuiting of the lesion and its later resection as a second stage (Harris, Bell and Brunn;[25] Fischer and Lürmann;[18] von Haberer;[24] Bell;[3] Galambos and Mittelmann;[20] Fenster;[17] Pemberton and Brown;[36] Ravdin and Rhoads;[38] Babcock[2]). Clute,[7] Homans and Hass[28] and Jackson[29] have suggested the wisdom of preliminary sidetracking with later resection if the symptoms are not relieved; but both Holm[26] and Estes[15] have emphasized the dangers of perforation or of profound toxemia with kidney and liver damage if resection of the short-circuited blind loop be too long deferred. Meyer and Rosi[32] suggest that the state of the mesentery of the involved bowel may be an indication as to whether to resect, short-circuit or leave alone. However, medical treatment seems to be futile in all true cases of the disease, and if instituted, seems only to allow time for further deterioration of the patient's resources of strength and resistance before surgical intervention becomes necessary.

The following two cases are presented as typical of moderately advanced regional enteritis, the second case by permission of the operator, Dr. Howard M. Clute. Although, as stated above, the disease predominates in young, Semitic males, both these patients were middle-aged, non-Semitic females.

Case 1. A 31-year-old, American woman was admitted to the hospital on April 2, 1936, with mucous diarrhea, pain in the right lower quadrant and occasional nausea and vomiting of 6 months' duration, poor appetite and a 24-lb. weight loss. Physical examination showed only emaciation, pallor and tenderness in the midepigastrium and right lower quadrant. The white-cell count was 9200, the red-cell count 4,100,000, and the hemoglobin 75 per cent. Urine examination, Wassermann, Mantoux and Widal tests and stool cultures were all negative. X-rays of the genitourinary tract showed an irregular filling defect of the cecum, constant at 5 and 24 hours, and loss of haustrations in the distal half of the colon, typical of colitis. An intravenous pyelogram showed a temporary delay in the lower right ureter, probably due to adhesions. For 2 weeks after admission the temperature varied from 98° to 101°F., the diarrhea, pain and tenderness and occasional vomiting continuing.

On April 16, at laparotomy by one of us (C. D. H.), the distal 30 cm. of ileum, the ileocecal valve and the proximal 7 cm. of cecum were resected, with end-to-side anastomosis of the ileum to the ascending colon. The postoperative course was uneventful and patient was discharged on the 19th day. Examination of the resected specimen showed very extensive ulceration of the mucosa of the terminal 20 cm. of ileum and of a small patch in the cecum. The bowel wall throughout this extent showed marked thickening and fibrosis, with infiltration by endothelial cells and lymphocytes. The lumen varied from 5 to 10 mm. in diameter. The serosa was injected and inflamed, and covered with fibrinous adhesions. Microscopically, the lesion did not resemble tuberculosis, and acid-fast organisms could not be demonstrated.

Twenty-one months later the patient reported that she had had no pains or vomiting, felt well, had three fairly well-formed stools daily, had gained 43 lb. since her discharge and was able to do her former work.

Case 2. A 45-year-old, Italian woman was admitted on February 18, 1937, complaining of sharp, persistent pain in the lower midabdomen for 7 months, and diarrhea, without blood or mucus, until 2 weeks before admission, since when she had been constipated. The appetite was poor, and there was a weight loss of 13 lb. in 6 months and

Figure 1. *Case 2. Roentgenogram taken 5 hours after barium meal and demonstrating all the characteristic roentgenologic changes found in regional enteritis: constant filling defect ("string sign"), proximal dilatation of ileum, and moth-eaten appearance of mucosa.*

64 lb. in 2 years. Physical examination revealed marked emaciation and pallor, and tenderness of the entire lower abdomen, with a movable, tender mass about 10 cm. in diameter just to the right of the midline, seemingly attached to the uterus. The white-cell count was 4650, the red-cell count 2,480,000, and the hemoglobin 70 per cent. Urine examinations, blood studies and Wassermann tests were negative. Stools on several occasions were strongly positive for blood.

After a preliminary 500-cc. transfusion, laparotomy was performed on February 24. Beginning about 15 cm. from the ileocecal valve and extending proximally for about 60 cm. the ileum was found to be markedly stenosed, thickened and edematous, with injection and inflammation of the overlying serosa. The adjacent mesentery also was thickened, firm and nodular. Only lateral anastomosis between the ileum and transverse

colon was performed, because of the extent of the lesion and the patient's cachexia. Convalescence was uneventful and the patient was discharged on the 16th postoperative day.

She was readmitted on April 20, about 6 weeks after the first-stage operation, feeling much better. A 5-lb. gain had been lost during the week preceding admission. Diarrhea and pains persisted, but were slightly less. Physical examination was essentially the same as on previous admission, but the red-cell count had risen to 4,900,000, with a hemoglobin of 80 per cent, and the stools were negative for blood.

On April 23, resection of about 40 cm. of ileum was performed with end-to-end anastomosis of the free ends of small intestine. Also, the ileocolostomy stoma, being found rather small, was enlarged according to the Mikulicz plastic technic. The temperature rose to 103.2°F. on the 4th postoperative day. Probing of the wound on the 8th day released much thin, fecal discharge, which continued thereafter. A barium enema done 27 days postoperatively showed a fecal fistula at the site of the ileocolostomy. The patient was discharged on the 30th postoperative day.

Eight months later, the patient reported feeling well with good appetite and no pain, nausea or vomiting. She had gained 40 lb. since leaving the hospital, but still had a very slight fecopurulent discharge from the lower angle of the incision.

SUMMARY

Regional enteritis is a disease to which no person is immune. Its symptomatology varies widely, including abdominal pain and tenderness, fever, leukocytosis, diarrhea with blood, pus and mucus, weight loss with anemia and asthenia, nausea and vomiting, abdominal distention and constipation. In any individual case any or all of these symptoms may be present and the tendency is toward progression, roughly in the order named.

In the individual case, symptoms can readily be correlated with the pathologic lesions, which may include stages of peritoneal inflammation, enteritis of varying severity, perhaps with abscess formation, obstruction of low or high grade, and fistula formation.

Histopathology is not helpful, and the etiology remains unknown, although many hypotheses have been suggested.

X-ray studies of the small bowel show that the "string sign" and mucosal changes are reliably diagnostic.

Optimal treatment in all true cases consists of resection of the lesion, in one or two stages.

Two typical cases are recorded, with clinical and pathological findings.

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THE PREVENTION AND CONTROL OF TUBERCULOSIS IN MASSACHUSETTS

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WE MEET to celebrate the twenty-fifth anniversary of the establishment of the Massachusetts Tuberculosis League.

Through the resignation of Dr. Francis P. Denny, vice president of the League since 1929, the Executive Committee has lost a wise counselor, experienced in the problems of public health, tuberculosis and the practice of medicine, and unselfishly devoted to the service of the community.

DEATH RATE IN MASSACHUSETTS

In Massachusetts, the death rate per 100,000 population from tuberculosis (all forms) has fallen from an annual average of 413 (1871-75) to 52.2 (1931-35), 43.1 (1936) and 42.7 (1937). The death rate from the pulmonary form of the disease dropped from 41.6 in 1935 to 39.4 in 1936 and rose to 39.9 in 1937.

IMPROVEMENT OF PUBLIC-HEALTH SERVICE

Attention was called at the last annual meeting of the League to certain recommendations by the Massachusetts Health Commission pertaining to the improvement of public-health service. One recommendation now under consideration is of special importance. To promote more effective control of diseases dangerous to the public health, standard procedures uniform for the State should be formulated. The act (House Bill No. 51) embodies desirable recommendations with respect to the control of these diseases and authorizes the State Department of Public Health to make such rules and regulations as it deems advisable for their control.

EXAMINATION OF SCHOOL PERSONNEL

In a few cities and towns there is some requirement with respect to health certificates from teachers, but this varies in the different communities and is for the most part confined to new teachers coming into the system.

The incidence of pulmonary tuberculosis among teachers may be estimated from figures collected from various parts of the country by Lees[1] at 2.15 per cent. It is, however, not to be expected that more than a fraction of these will be found to have open pulmonary tuberculosis.

The hazard to children from tuberculosis in teachers is much the same as that with the disease in the family, and the program in the State should be extended to include this aspect of the problem. It would, in consequence, be desirable to adopt a uniform requirement, such that no person with active pulmonary tuberculosis or any form of tuberculosis which may endanger the health of children be permitted to be in contact with them.

INVESTIGATION OF SCHOOLCHILDREN

In most of the communities in the State the schoolchildren are routinely examined for tuberculosis. In Boston, during the past year, an examination was made of children in the parochial schools, and plans have been formulated to investigate the children in the public schools.

In the examination of schoolchildren for tuberculosis, it has seemed desirable to change from the annual investigation of the seventh, ninth and eleventh grades to that of the high-school group as a whole. This change eliminates repetition of the examination of two grades after the lapse of two years. It has the further advantage of enabling the examiners to study a larger number of children in one school at one time, and to return to the school when other children have taken the place of those already examined. A larger proportion of children with tuberculosis is to be expected in the high schools than in the grade schools.

RESULTS OF THE EXAMINATION OF HIGH-SCHOOL CHILDREN

Owing to the usual absence of significant symptoms and signs with tuberculosis in childhood, it is only by routine investigation that the disease can be discovered. From the experience with the tuberculin and x-ray method in the Chadwick Clinics among high-school children, it may be expected that at least about 1 in 80 will be found to have the childhood type of tuberculosis, and about 1 in 700 the adult type.

The advantages to be derived from the finding of tuberculosis in schoolchildren are twofold. There is on the one hand the benefit to the affected child and on the other the benefit to the community. These advantages are, however, fully realized only when the investigation includes both children and contacts.

In the investigation of the high schools, the finding of at least 8 cases of the childhood type to 1

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of the adult type makes the former the most important group numerically.

From the point of view of seriousness, however, the two forms of the disease are quite different. The childhood type is a first infection. The disease is usually mild and the outlook favorable. Its discovery is to be regarded as a safeguard in the opportunity offered to eliminate further infection from without, for instruction regarding exercise, rest and diet and for further x-ray examinations. It is to be expected that by these means further progress will be prevented in many cases, with consequent saving of life and the elimination of an expensive illness. On the whole, however, in consequence of its usual benign course, the most important advantage to be derived from the discovery of the childhood type is the opportunity afforded to find and eliminate a source of infection through the investigation of family contacts.

The pulmonary form of tuberculosis, the adult type, is serious, and the outlook in children is poor. Its discovery affords the opportunity for the institution of treatment, the elimination of a source of danger to others by removal from school and the examination of family contacts.

It is apparent that one of the most important results of the investigation of schoolchildren for tuberculosis is the opportunity afforded to trace the disease to its source in the family and to eliminate further infection of the child and others by the breaking of contact.

COST OF THE EXAMINATION OF SCHOOLCHILDREN

The expense of the school program is estimated by Zacks[2] as approximately $1.50 per child tested with tuberculin. On this basis, inasmuch as about 80 high-school children have to be examined to find 1 with the childhood type of tuberculosis, the cost for the discovery of this form of the disease is $120 per case. To find the adult type in 1 child out of 700 costs about $1000 per case.

From the financial point of view, however, it seems uneconomical to spend this amount on the discovery of the disease in children and not make use of the opportunity it affords to extend the investigation to the probable source of infection in members of the family in all cases.

EXAMINATION OF CONTACTS OF THE TUBERCULOUS

Case-finding is most successful among the family contacts of known cases, and an x-ray examination should be made of all household and other contacts of tuberculous patients in the practice of physicians and in the state, county and municipal sanatoriums, and of all schoolchildren found to have tuberculosis. Facilities are fortunately available for the x-ray examination of indigent patients and contacts through outpatient departments of the state and county sanatoriums and the extramural diagnostic clinics.

The importance of the investigation of contacts of sanatorium patients is illustrated by the results in 600 members of the families of 144 patients with active pulmonary tuberculosis at Middlesex County Sanatorium and Westfield State Sanatorium. Pope[3] reports that 46, or 7.7 per cent, were found to have pulmonary tuberculosis. A recent survey by him indicates that extension of the examination to include family contacts of sanatorium patients is now carried out in a large proportion of cases.

Extension of the investigation to include the famil, contacts of schoolchildren with the adult and childhood (hylum) type of tuberculosis is also desirable. The proportion of family contacts of tuberculous school children thus far examined is far too low, and in certain communities does not exceed 10 per cent.

The importance of the examination of family contacts of children with the adult type of tuberculosis is illustrated by the results in 6 cases. Zacks[4] reports that in the families of these children the examination of 30 persons led to the discovery of 6 additional cases of the adult type of pulmonary tuberculosis, or 20 per cent in the group, and 1 hylum case.

The importance of the examination of the family contacts of children with the childhood type of tuberculosis is also emphasized in the findings by Zacks. In 89 hylum cases 85 families, including 346 additional persons, were examined. In this group, 14 cases (4 per cent) with pulmonary tuberculosis or suspicious lesions and 12 additional cases of the childhood type were found.

Failure to extend the investigation of high-school-children with the childhood type to include family contacts, in whom about 1 in 25 may be expected to have pulmonary tuberculosis, deprives the individual and the community of one of the most important advantages of the project.

It is obvious that the finding of one individual with tuberculosis should be regarded only as a first step leading to an investigation of an infected family.

Not only is the proportion of those with the disease much higher among contacts than in the population at large, but the disease is likely to be discovered at an earlier stage. Owing to the lack of significant initial symptoms it is not to be expected that persons with incipient pulmonary tuberculosis will seek medical advice. A larger proportion of cases without extensive lesions and capable of arrest under treatment is to be expected in contacts than in other groups. The breaking of contact protects the family and the community.

Extension of the investigation to include family contacts has an important economic aspect. The investigation of schoolchildren and the provision and maintenance of sanatoriums for the care of the tuberculous are expensive projects. The sanatoriums not only help the affected individual but serve through segregation of the tuberculous to protect the family and the community. The discovery of the childhood type of the disease in the schools and the investigation of family contacts may be expected to prevent further infection by the breaking of contact and to halt the progress of the lesions to a stage where institutional care of the individual becomes a charge on the community.

In communities in which the family contacts of sanatorium patients have already been investigated, it is found that in many instances the contacts of tuberculous schoolchildren have already been examined.

HEALTH EDUCATION

There is need for improvement in health education in the schools, and especially in the instruction regarding the prevention and control of tuberculosis.

Tuberculosis still remains a leading cause of death in the teens and the early twenties, and parental consent for the examination of high-school children is obtained in far too small a proportion of cases. It is to be expected that more systematic teaching on the subject of tuberculosis will result in a larger number of consents. Tuberculosis teaching should be carried out in the grades to be examined about a month prior to the tuberculin testing of the children. Material prepared by the Massachusetts Tuberculosis League is available. The teaching should be completed before the consent slips are sent to the parents.

After enlisting the co-operation and support of the school superintendent and principal in the project, someone well informed on tuberculosis, preferably the physician in charge, should explain the purpose and importance of the examination at a meeting of the teachers.

At the time the consent slips are distributed in any school, it is desirable for the physician in charge of the examination to outline the project to the assembled children. The film "Behind the Shadows," prepared by the National Tuberculosis Association and designed for high-school pupils, may well be shown at the time. The League film, "Preventing Tuberculosis," is also available.

It is desirable that the school nurse visit the houses to obtain consent from the parents who have refused to have their children tested.

REHABILITATION

The National Tuberculosis Association has made a survey of the occupational experience and ambitions of sanatorium patients. A separate Division of Rehabilitation Service has been established. In collaboration with the Federal Vocational Rehabilitation Service and some of the state rehabilitation services and the state tuberculosis associations, field studies have been launched to determine the proportion of discharged patients who have died, gone back to work or are unemployed.

Sanatorium facilities in Massachusetts provide physical care for patients with tuberculosis during their illness, but the problem of the individual after discharge has been largely neglected.

The Boston Tuberculosis Association, however, conducts an important rehabilitation project, the Sheltered Workshop. It employs patients in whom the disease is quiescent or arrested. During 1937, 40 patients under trained supervision were provided with such work as hand and machine sewing for women, and woodwork, upholstery and woodfinishing for men. Returns from sales are insufficient to meet the cost of wages and other expenses, and the deficit is met from a private source.

In the summer of 1934, at the invitation of Dr. Sumner H. Remick, representatives of the National Tuberculosis Association conducted aptitude tests and gave educational guidance to a selected group of patients at the Middlesex County Sanatorium. During the fall of 1934, the Executive Committee authorized the expenditure of league funds for two courses, one in business psychology and the other in household management.

Plans are being made for the reorganization of the Central New England Sanatorium and Industrial Colony, and it is to be hoped that a rehabilitation center for the tuberculous can be developed at the institution.

Limited experience in Massachusetts and elsewhere indicates that the problem of rehabilitation on any large scale will be expensive. Considerable initial investment is needed to launch the enterprise, and an endowment or subsidy is necessary to maintain it. Restoration to economic independence in full or in part and fewer relapses after return to work may, on the other hand, be expected to lessen the financial burden on the community.

The Executive Committee recognizes the importance of rehabilitation, and plans to take part in helping to solve the problem.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24311

PRESENTATION OF CASE

A fifty-two-year-old, white, Canadian widow entered the hospital with the complaint of abdominal pain of five and a half hours' duration.

A few hours following a very large Thanksgiving dinner she began to have very severe, steady, upper abdominal pain accompanied by nausea and frequent vomiting. Two and a half hours later a physician was called and gave her morphine (¼ gr.). Her abdomen was not spastic or rigid at that time. However, the symptoms persisted; and two hours later, one hour before admission, the pain was much more severe, and the abdomen was distended and spastic throughout. The nausea and vomiting continued unchecked.

For several years she had suffered from indigestion and "gas on her stomach" and had occasionally had attacks of mild upper abdominal pain. She had never been jaundiced. Because of her extreme illness a satisfactory past history could not be obtained.

Physical examination revealed a thin, poorly nourished, somewhat cyanotic woman with cold skin. She was apparently suffering from waves of nausea, climaxed by weak vomiting of large quantities of pink, sweet-smelling fluid. She seemed to be in extremis. The pulse was almost imperceptible, and the heart sounds were scarcely audible. The blood pressure could not be obtained. The lungs were clear. The abdomen was tremendously distended and tight as a drumhead. Even the perineum was bulging. The percussion note was dull in the flanks and tympanitic elsewhere. There was very marked muscular spasm, but there was no great amount of tenderness, probably due to the patient's weakened condition. No peristalsis was audible. There was slight pitting edema of both lower legs.

The temperature was 95°F., the pulse 50. The respirations were 23.

The white-blood-cell count was 12,200. X-rays of the abdomen showed tremendous dilatation of the stomach, colon and small bowel. The lateral view taken with the patient lying on her back demonstrated a wide space between the dilated loops of bowel and the abdominal wall, apparently due to large amounts of free gas in the peritoneal cavity.

Shortly after entry an abdominal paracentesis was performed. When the trocar entered the abdominal cavity, sweet-smelling gas escaped under great pressure. Only a portion of the gas was allowed to escape and a clamped catheter was left in place. A No. 10 stomach tube was put down, and Ochsnerization begun; only small amounts of gastric fluid and partially digested food were obtained. She was given a transfusion with little improvement. Four hours after the paracentesis the catheter clamp was released, but only a small amount of air and thin yellow fluid escaped through it. She died quietly two hours later. Shortly before death the abdomen was still tremendously distended, and peristalsis absent. No gas had been passed by rectum.

NOTES ON THE HISTORY

DR. GEORGE A. LELAND: "A few hours following a very large Thanksgiving dinner she began to have very severe, steady, upper abdominal pain accompanied by nausea and vomiting." That story is suggestive perhaps of overindulgence; and the type of nausea, vomiting and pain is characteristic of a person passed middle age who has had indigestion perhaps of the biliary type, with the results as noted.

The fact that morphine was given suggests that the pain was very severe, and one thinks, of course, of real colicky pain. Such pain should be relieved by a quarter grain dose, whether it is colic of the biliary tract, of the renal tract or of the gastrointestinal tract.

There is no note of any tenderness, so we do not have any localization to suggest where this individual's trouble started. It is interesting that the abdomen was not spastic or rigid when the hypodermic was given, and in spite of this the pain persisted and was severe enough to give spasticity a few hours later.

The past history furnishes a sort of confirmatory background that she had had indigestion and that this overindulgence at Thanksgiving had caused an exacerbation of her difficulties. We are inclined perhaps at this point to think of her having something more severe than just plain biliary colic.

"Physical examination revealed a thin, poorly nourished, somewhat cyanotic woman with cold skin." We shall point out that a thin, poorly nourished woman might indicate that there had been loss of weight, even though no such history was obtained.

"She was apparently suffering from waves of nausea, climaxed by weak vomiting of large quan-

tities of pink, sweet-smelling fluid." That would indicate some overwhelming condition which caused very definite abdominal shock. The last phrase is intriguing. She had had Thanksgiving dinner, and one can think of all sorts of things pink — tomato juice, cider, cranberry juice and various forms of alcoholic beverages, such as claret lemonade and punches. This color might have had something to do with bleeding, but the significant point here, I think, is the sweet-smelling fluid.

"She seemed to be in extremis. The pulse was almost imperceptible, and the heart sounds were scarcely audible." That group of signs of course goes with cyanosis and the cold skin. Some tremendous, overpowering, shocking situation had arisen. The fact that the lungs were clear in spite of the imperceptible pulse and the absence of blood pressure likewise indicate something very rapid and overwhelming, otherwise we should expect rales — a wet lung of some sort.

"The abdomen was tremendously distended and tight as a drumhead. Even the perineum was bulging." That is a combination which I have not come across since obstetrical days. In my experience it is indicative of the second stage of labor.

"There was slight pitting edema of both lower legs." That might have been pitting edema of a chronic nature. I am rather inclined to think it was, because she had no signs in her lungs of cardiovascular failure.

The pulse of fifty indicates something very profound in the way of shock or something in the heart that gave her a block. It is obvious that her lungs were all right. Her respirations were still low, and we should not expect a dose of morphine, so small that it did not relieve pain, would have any depressing effect on the respiratory rate.

"The white-blood cell count was 12,200." Just enough to be a teaser. The blood did not react to any appreciable degree; it was just a little above normal. X-rays of the abdomen were helpful. There we find something that tells us a lot, in fact perhaps accounts for the absence of tenderness, in spite of great spasticity. I recall a patient who was blown up with air following a gastroscopy, in whom not only the gastric cavity but also, by perforation, the abdominal cavity had been inflated. There was no localized tenderness: the air acted as a cushion between the abdominal wall and the gastric wall, thus screening any tender point. Of course the presence of this amount of free gas absolutely indicates perforation of some hollow viscus, and however much we might have thought of the biliary system as being responsible for this situation, the presence of gas definitely rules it out. So we can assume that we are dealing with perforation of a hollow viscus. Whether it is the stomach, intestine or colon, we have not as yet positively determined.

We still have this sweet-smelling liquid. I cannot imagine that tomato juice, cider or cranberry juice mixed up in a turkey dinner with added gastric juices would still be sweet-smelling when vomited or that gas derived therefrom and removed from the abdomen through a trocar opening would have such an odor.

DR. HENRY H. FAXON: May I interrupt? There are several things that are not mentioned in the history which may be of some help. In the first place the vomitus showed blood on guaiac test. Secondly, when her physician first saw her the abdomen was said to be not distended and in the course of two hours apparently all the distention present at entry had taken place. Thirdly, for what it is worth, she had taken for her gastric distress large amounts of sodium bicarbonate, in addition to the morphine.

DR. LELAND: Which, of course, would give her still more gas.

"Only a portion of the gas was allowed to escape, and a clamped catheter was left in place." That is interesting too. I assume that the reason for that procedure was that the surgeon in charge thought that by avoiding complete deflation of the abdomen enough pressure might be left intra-abdominally to prevent the escape of a great deal of contents from the perforation.

DIFFERENTIAL DIAGNOSIS

DR. LELAND: In summary of this case it is quite obvious that an individual in chronically poor condition comes into the hospital with some acute surgical emergency of the abdomen that has been very overwhelming and has rendered her practically moribund on entrance, and accordingly, no matter what the diagnosis, very little hope could be entertained for getting the patient well. Therefore, supportive treatment was given in the forlorn hope that Ochsnerization and transfusion would improve her condition enough to permit surgical intervention. But, as we see, these measures failed, and the patient died.

It is interesting to venture some impressions as to why she died, what she died with and what she died from. We find an individual of fifty-two who was thin and poorly nourished, with indigestion for several years. Indigestion at that time of life may be due to a number of things, but combined with the age and the poor condition

of the patient, we are a little bit inclined to think of cancer, or at least we think that it should be ruled out. This individual overtaxes her stomach by a tremendous meal, apparently, and begins to have symptoms which are atypical of perforation. She does not have a sudden, sharp abdominal pain, but a few hours after eating she begins to have a severe, steady pain. When ulcers perforate, they usually call attention to that fact by an acute stabbing type of pain. Of course there are exceptions to every rule, but 90 per cent of them act in this way. On the other hand, the symptoms of cancers that perforate are apt to be atypical, in that the pain is less pronounced and is recorded oftentimes as being just as described here—a steady, increasing pain. I think we can assume that the perforation was of the stomach—the sweet-smelling gas would thus be accounted for. At least the gas was not foul smelling, hence ruling out perforation of the bowel.

I am taking the stand that she had a malignant tumor of the stomach—a carcinoma which perforated because of the overdistention which followed overeating. Sometimes cancer patients in whom perforations of the stomach occur develop tremendous and overwhelming sepsis and die much more quickly than those with perforated ulcers. So it is not inconsistent with the duration of the story for her to have died from septic peritonitis due to a perforated malignant tumor of the stomach. On the other hand there are other factors about this case which I think are very intriguing; they all devolve upon the extreme shock that she was in and this sweet-smelling fluid and sweet-smelling gas.

A number of years ago I saw a woman die from acute alcohol poisoning following the inadvertent introduction of 70 per cent alcohol into her peritoneal cavity. Following curettage for incomplete abortion in a case in which the uterus had been ruptured, an alcohol douche poured into the peritoneal cavity. This was so long ago that I do not distinctly remember all the details, but I know that the case was quite confusing from the vasomotor point of view. She had cyanosis, a slow pulse and imperceptible blood pressure, and died within six or eight hours of her surgical accident. In reading over this history I am reminded of that case, and so I believe that here we might account for the cyanosis, the tremendous shock to her cardiovascular system and the rapidly fatal outcome, not necessarily by peritonitis, but by escape of alcohol from the stomach into the peritoneal cavity.

Alcohol peritonitis might perfectly account for a pulse of fifty, but that is not the only possible explanation; she might have had cancer or one of the bizarre fibroblastic tumors with metastases to the heart or even to the bundle of His. My final diagnosis is carcinoma of the stomach with perforation and with rapid dissolution due to the escape of alcohol into the peritoneal cavity.

X-Ray Interpretation

Dr. Aubrey O. Hampton: This is the usual flat abdominal film showing a markedly dilated stomach and many gas-filled loops of dilated bowel below it. The entire abdomen appears more radiant than normal. In the lateral view with the patient on her back you see a remarkable appearance. The abdomen looks like a large balloon filled with many smaller balloons.

Clinical Discussion

Dr. Faxon: The unbelievable degree of distention was the thing of primary interest in this case. One would have had to see the patient to appreciate it, and even the x-ray film brings out only in part the degree of distention that this woman had. Despite the astonishment that Dr. Leland showed about the bulging perineum, I can assure you it was a fact. How she could have blown herself up to such a degree was a mystery to us. Whether the sodium bicarbonate played a part is one of the things we speculated about. We were certain that she would not stand major surgery and that her shock, slow heart rate and other signs of being profoundly ill were due in large part to this extreme degree of distention. The reason for slowly decompressing her was not that the stomach would leak less but that sudden deflation would precipitate her death. A small caliber needle was put into the lower abdomen, and so great was the pressure that when the syringe was put into it the plunger shot out. We thought we could slowly decompress her, and if she did rally, we could then do something further.

There is one other point of practical importance: when she first came in vomiting small amounts and retching we thought it would help her if we passed a stomach tube and deflated the stomach, as is customary. The small type of tube was passed. It was irrigated back and forth, and in the period we observed her, we were convinced that we had deflated the stomach. It was rendered perfectly obvious by subsequent events that this tube did not begin to cope with the amount of distention present in the stomach itself because the small lumen of the tube plugged too easily to permit free drainage. Had a tube of sufficient caliber been used, the stomach could have been satisfactorily emptied. We considered the possibility of having Dr. Benedict peritoneoscope the

patient, not only to deflate the peritoneal cavity but also to see if the site of perforation could be determined, however the delay which would be entailed in carrying out this idea seemed unjustifiable. We had the flat x-ray plate taken primarily for teaching purposes, as we were sure of the diagnosis but believed that a film of such an outstanding degree of abdominal distention should be on record. We were misled by the use of too small a tube, as already pointed out, in thinking we had deflated the stomach and that most of the air was free in the peritoneal cavity. It is true that there was a large amount of air in the peritoneal cavity at the time of autopsy, but it is also true that a great deal of gas was still present in the stomach.

Clinical Diagnoses

Perforation of upper gastrointestinal tract.
Probable carcinoma of stomach with generalized peritonitis.

Dr. Leland's Diagnoses

Carcinoma of stomach with perforation.
Alcoholic poisoning from peritoneal absorption.

Anatomical Diagnoses

Acute gastric dilatation.
Acute hemorrhagic gastritis.
Acute ulceration of the stomach with perforation.
Pneumoperitoneum.
Acute generalized peritonitis.

Pathological Discussion

Dr. Tracy B. Mallory: I read a detective story years ago in which the murder was consummated by giving an individual the two fractions of Seidlitz powders in two, closely consecutive potions of fluid. At the time I was very skeptical as to whether death could really result from such a procedure. At the wind up of this case I am inclined to believe that the idea was not entirely absurd and that some similar mechanism may have been the cause of this patient's death. The findings at autopsy were those of an extreme degree of gastric dilatation with a generalized almost hemorrhagic gastritis. There were multiple acute ulcers, one of which had perforated through the stomach wall. The abdominal cavity contained tremendous amounts of gastric contents. We did not make any test for alcohol and to our noses the aroma was not particularly suggestive of alcohol, but I cannot rule that out. This is a color-photograph of the stomach which will allow you to judge for yourselves the extensiveness and the fiery redness of the diffuse hemorrhagic gastritis.

Some of you may remember a case of Dr. Simeone's, discussed within a couple of weeks, in which an intestinal obstruction from an adhesive band was allowed to run untreated for a number of days before the patient came into the hospital, and in which quite enormous distention of the small bowel was present. The patient subsequently developed diffuse hemorrhagic enteritis of the portion of jejunum previously distended. That case and this one raise the question as to whether it is not possible to distend an abdominal viscus so much that the blood supply is temporarily, completely blocked and necrosis initiated. Cultures and sections of the stomach showed no streptococci, so that a phlegmonous process can be ruled out. I think it is certainly possible, even probable, that the primary cause of death was acute gastric dilatation and that the ulcers were secondary and not primary.

CASE 24312

Presentation of Case

A fifty-two-year-old, white, American housewife entered the hospital with the complaint of pain in the back for three months.

The patient was entirely well until one year before entry when she began to complain of stiffness in both legs which she described as "rheumatic." Associated with the stiffness were some weakness and clumsiness. She also occasionally dropped dishes. Three months before entry she began to have a constant, dull, sometimes burning, gradually increasing pain in her low back, which was more marked on the right side and was worse in the morning. She began to lose weight, strength and appetite, and noticed tingling sensations in both legs. Strapping her back gave no relief. She found that the most comfortable position she could assume was lying on her back with her feet and head higher than her body. Two months before entry she entered an outside hospital and remained there until just before entry to this hospital. While there the pain began to radiate to her legs and became even more severe so that she had to be assigned to a private room because of her constant groaning, which disturbed the other patients. She did not have numbness, other paresthesias or any apparent weakness; nor did she have any other symptoms such as nausea, vomiting, headache or mental derangement of any kind. Three lumbar punctures were done at the outside hospital, all of which showed normal dynamics. The spinal fluid,

one month before entry to this hospital, showed a cell count of 4 polymorphonuclears per cubic millimeter, a total protein of 50 mg. per cent and a negative Kahn. One week later it showed a cell count of 5 polymorphonuclears and a total protein of 85 mg. per cent. Two and a half weeks later, 5 days before entry, the spinal fluid was yellow and clear, and showed a total protein of 500 mg. per cent. The cell count was 3 polymorphonuclears and 14 lymphocytes but was thought to be inaccurate because of clotting of the fluid. Urine specimens taken by ureteral catheter showed on the left 50 or 60 red cells, 4 or 5 white cells and a few casts per high-power field; and on the right, rare red cells and white cells and many granular casts. X-rays taken at the outside hospital showed a normal chest, normal retrograde pyelograms, sacralization of the transverse processes of the fifth lumbar vertebra, mild hypertrophic changes of the dorsolumbar spine and sacroiliac joints and complete bilateral calcification of the iliopsoas band. During her illness she lost 15 to 20 lb., having previously weighed about 200 lb.

Her mother died of cancer of the stomach, and two sisters died of cancer of the breast, one of them with metastases to the spine.

Physical examination revealed a well-developed and nourished woman complaining of severe pain in her back. The right pupil was smaller than the left, but both reacted to light. The lungs showed coarse bronchitic rales over the sternum and questionable increased breath and voice sounds at the left base. There was a soft systolic heart murmur to the left of the sternum. The blood pressure was 180 systolic, 110 diastolic. The abdomen was negative. The left ankle jerk was somewhat more difficult to obtain than the right, but the extremities were otherwise negative. There was a red maculopapular rash over the back. A pelvic examination was negative.

The temperature was 101°F., the pulse 102. The respirations were 25.

The urine had a specific gravity of 1.035 with a slight trace of albumin but was otherwise negative. The blood showed a red-cell count of 3,650,000 with 65 per cent hemoglobin and a white-cell count of 14,500 with 63 per cent polymorphonuclears. The stained blood smear showed 2 nucleated red cells per 100 white cells. The nonprotein nitrogen of the blood was 31 mg. per cent, and the total protein 5 gm. per cent; a Hinton test was negative. A lumbar puncture showed apparently normal dynamics. The spinal fluid was clear and xanthochromic and had a cell count of 2 lymphocytes, a total protein of 858 mg. per cent, a sugar of 54 mg., a chloride of 658 mg., a negative Wassermann and a 5555554444 goldsol curve. The fluid coagulated after fifteen minutes.

An x-ray of the chest showed diffuse mottling of the upper two thirds of the left lung field, with marked thickening of the apical pleura on the left.

A neurological examination on the third hospital day revealed a right sixth-nerve palsy, bulbar speech and suggestive Kernig signs. There were no sensory changes. Mentally there was some confusion and disorientation. The white-cell count on that day was 39,700, and her temperature had risen to 104°F., with the pulse 160 and the respirations 60. On the following day a lumbar puncture showed an initial pressure of 130 mm., with normal dynamics. After 10 cc. of fluid had been removed the pressure was 0. The spinal fluid was clear and pale yellow and had a cell count of 2 lymphocytes and 3 red cells, a total protein of 726 mg. per cent, a sugar of 59 mg. and a 0000001222 goldsol curve. On the fifth day she developed a left facial weakness of the central type, and on the following day she died.

Differential Diagnosis

Dr. George C. Caner: It seems to me that it would be worth while to discuss some of the individual symptoms before trying to bring them together and attempting to make a diagnosis. In the first place we have a history of stiffness in both legs, weakness and clumsiness, and a tendency to drop dishes going back a year. We are not told whether these symptoms continued up to the day of entry, and they do not seem very definite. I am skeptical as to whether they are related to the disease process which led to her death, particularly since there are no neurological findings to account for them.

Three months before entry, she developed a severe, constant, dull, burning pain in the back which became progressively worse. It was worse in the morning and she felt more comfortable lying in the position which was described. The latter characteristics suggest an arthritic process and the x-rays show some hypertrophic changes, but it seems to me that the pain was much too severe, steady and progressive to be explained on that basis.

Later she began to have tingling sensations in both legs, and the pain radiated to her legs and was very severe. These symptoms indicate a neuritic involvement due either to extra- or to intraspinal disease. It was probably because of the possibility of an intraspinal lesion that the lumbar punctures were done. The first lumbar puncture gave a fluid containing 4 polymorphonuclear leukocytes and a

slightly elevated protein (50 mg. per cent). The 4 polymorphonuclears are of course abnormal. In subsequent examinations leukocytes were also reported, so that one must consider them seriously. However, they were not present in the examinations done at this hospital, so that I am somewhat skeptical of them. The most striking thing in the lumbar-puncture findings is the high total-protein content. At the second admission the total protein was 85 mg. per cent, two and a half weeks later it was 500, and still later, subsequent to entry to this hospital, it became even higher. It therefore looks as though the total protein had increased very rapidly: this is an unusual and interesting finding. It is possible, however, that such was not the case. If the first lumbar punctures were done at a high level and the subsequent lumbar punctures were done at lower levels and if a block were present between these levels or even below them, the differences in the amount of total protein might be explained without assuming that the total protein in the fluid had rapidly increased.

The most frequent cause for increase in the total protein of the spinal fluid is a blocking of the spinal-fluid pathways. If a block is present we must consider at what level we are to expect it and what type of lesion may be causing it. The fact that there were so few cells and such a high total protein suggests very strongly that tumor rather than some inflammatory condition is the cause of the block, if any exists. There is no evidence of compression of the cord, and if the increased protein were due to a block high enough to cause cord compression one would expect to find evidence of involvement of the sensory or motor tracts. If a block were present one should therefore expect it to be below the level of the cord, involving the nerve roots of the cauda equina. A tumor at this level involving nerve roots could cause marked and very severe pain without giving rise to diminished sensation or to motor symptoms. The only evidence of block, however, is that at the last lumbar puncture, done just a few days prior to death, the pressure fell from 130 mm. to 0 after the removal of only 10 cc. This suggests that the fluid was removed from a small reservoir, but this evidence does not seem to me to be sufficiently conclusive of block. It is possible that something obstructed the needle after 10 cc. had been removed. In view of the fact that the dynamics were repeatedly found to be normal, it seems unlikely that the high total protein was due to a complete spinal-fluid block. We therefore must consider other possibilities.

One sometimes finds a high total protein in cases of brain tumor. Usually it is not over 400 mg. per cent, but it may rarely be as high as it became in this case. A high total protein is particularly common in tumors involving nerve roots in the posterior fossa. There were no symptoms or signs of increased intracranial pressure in this case. Of course their absence does not rule out brain tumor but does make tumor less likely. Even the late neurological symptoms could not be explained by a single large tumor. They can only be explained by multiple lesions, so brain tumor seems unlikely unless there were multiple tumors.

One observes a high total protein in cases of infectious polyneuritis of the type that often gives facial diplegia; but since this disease causes marked weakness rather than severe pain it seems to me we can rule it out. One may find a high spinal-fluid protein in cerebral hemorrhage, but, if so, one would expect red cells, and there were no red cells in the fluid. One might get it in cerebral infarct, and in this case there might not be any red cells. So much for the consideration of the spinal fluid: it leaves us still somewhat perplexed as to what might be causing the high total protein.

The finding of red blood cells and of casts in the urine is evidence of lesions in the kidneys which we must try to explain. The fact that the patient lost 15 to 20 lb. in a short time and that she had an anemia indicates that she had a debilitating disease. One therefore has to think of some debilitating disease which would cause the spinal-fluid findings and which would also cause multiple focal lesions. Intraspinal or spinal disease is suggested by the pain in the back. Apparently there was abnormality in the kidneys, and later on, just before death, she developed multiple foci in the brain and in the lungs.

Of the debilitating diseases that might cause multiple focal lesions, one thinks of tuberculosis, syphilis, sepsis, cancer and subacute bacterial endocarditis. The spinal-fluid findings are greatly against tuberculosis, syphilis or sepsis. None of these would give such a high total protein and so few cells. One comes then to the possibility of malignant tumor. There is a suggestion of this in the fact that there is such a striking history of cancer in the family. Cancer would explain a good many of the symptoms. It would explain the debilitating symptoms. It might explain the x-ray findings of mottling in the chest. It would explain the anemia and also the pain in the back, if there were metastases to the spine. X-rays in the earlier stages of the disease might well be negative. There are no symptoms or findings of importance that cancer cannot explain and there are no symptoms or findings that make it unlikely.

The fact that she had a systolic heart murmur

makes one think of the possibility of subacute bacterial endocarditis. That would explain a good many of the symptoms of the case. If there were multiple infarcts in the brain it might account for the spinal-fluid findings and the neurological signs that she developed shortly before death. Infarcts could well explain the mottling in the lungs, and the urinary findings are rather characteristic of subacute bacterial endocarditis. However it would not cause severe pain in the back and legs. One would have to have some other lesion to explain these symptoms. I think, therefore, that subacute bacterial endocarditis is unlikely.

One might entertain the possibility of chronic nephritis with hypertension. There was evidence of a nephritic abnormality in the urinary findings, but a diagnosis of chronic nephritis does not seem warranted. The kidney function was not impaired. This diagnosis also leaves the pain in the back and legs unexplained.

I believe that we might be helped by the x-rays.

DR. TRACY B. MALLORY: There is no x-ray man present. Dr. King, would you care to express an opinion?

DR. DONALD S. KING: I cannot make out much in these films except that there is the mottling spoken of. I think it would be difficult to make a definite diagnosis.

DR. CANER: Do the changes suggest metastatic cancer?

DR. KING: No; to me, they do not. I do not recall that we have had a case of metastatic cancer with lesions limited to one chest like this. The mottling could be, of course, due to bronchopneumonia or tuberculosis; but it is not characteristic of tuberculosis.

DR. CANER: We might be helped a good deal by looking at the temperature chart. May we see it?

DR. MALLORY: The chart is not very revealing. She came in with a temperature of 101°F.; it rose after two days to 104°F. and continued elevated until she died.

DR. CANER: Do we know what it was in the other hospital?

DR. MALLORY: It was normal.

DR. CANER: This is against an infectious process, such as a subacute bacterial endocarditis or sepsis, and more in favor of cancer. I suppose that prior to death she developed a bronchopneumonia. The best diagnosis I can make is metastatic cancer with a terminal pneumonia. I see nothing to indicate what type of malignant disease it was or where it was primary but believe that it caused the pain in the back by involving nerve roots and that there were metastases in the brain and in the kidneys and lungs.

DR. MALLORY: There is one rather unusual finding recorded in the laboratory work; in spite of the relatively mild secondary anemia, 3,600,000, nucleated red cells were seen in the smear. Have you any suggestions about that, Dr. Richardson?

DR. WYMAN RICHARDSON: I think that it would go with any widespread, malignant-tumor involving the bone-marrow. It would indicate that there was not simply a single or just a few focal areas, but a very widespread involvement of the bone marrow. One would like to see skull plates and better plates of the pelvis. I do not believe that it helps in regard to the discussion as to what kind of tumor was present.

DR. HARRY C. SOLOMON: From the picture, as it is given here, you have a very confusing story. I believe that one could do no more than speculate on the presence of cancer. Such a diagnosis is always a good bet in a patient who dies at about this age with a widespread disorder. If one were dealing with a detective story one would say that it is too obvious. The fact that the two sisters had malignant disease makes it seem too probable; so one would tend to look for something less likely. However, this is the type of reasoning one uses in solving a detective story, not in diagnosing a medical case.

The blood picture and neurological findings certainly are suggestive of some type of cancer. I still do not know why she should have had a high protein in the spinal fluid without evidence of block. If one were dealing with the hypothesis that she had widespread cancer,—as we think she must have,—there should be some signs in the few bones of which we have x-ray pictures. It is rare to get cancer of the spine with high protein without changes in the bones themselves. I do not find any specific evidence to make me think of lymphoblastoma, yet that is what I should have to think of as the first possibility, based on the general picture. Secondly, I am wondering if she did not have an infectious polyneuritis. That might explain the high protein and the diffuse neurological signs. While the case is far from typical, that would be my second diagnosis.

DR. RICHARDSON: The blood picture is definitely against infection.

DR. SOLOMON: Yes, but so-called polyneuritis does not show the systemic picture of general infection.

DR. CANER: She had a central rather than a peripheral facial weakness. In an infectious polyneuritis the facial paralysis that often occurs is, so far as I know, always peripheral. If she had had an infectious polyneuritis at the time she had the high total protein, she ought to have had weakness and perhaps some impairment in sensation;

furthermore, the severe pain would have been very unusual.

DR. SOLOMON: That is logically correct, but there are a great many variations that come up so that one does not like to make a hard and fast rule.

CLINICAL DIAGNOSES

Carcinomatosis?
Tuberculous meningitis?
Bronchopneumonia.

DR. CANER'S DIAGNOSES

Metastatic tumor, ? source.
Bronchopneumonia.

ANATOMICAL DIAGNOSES

Primary fibrosarcoma of the dura with metastases to bones, lungs and adrenals.
Bronchopneumonia, slight.
Cholecystitis, chronic.
Cholelithiasis.
Parovarian cyst.

PATHOLOGICAL DISCUSSION

DR. MALLORY: At the time of autopsy we found that there was tumor present in many parts of the body. The major focus of tumor which was seen was in the dura of the spinal cord, running from the midthorax region down to the cauda equina. The dura overlying the anterior surface of the cord was thickened up to several millimeters throughout this distance. The tumor did not extend into the underlying vertebrae. We also found metastatic foci of tumor in the adrenals and scattered throughout all the bones of the body—sternum, ribs, and long bones as well. We made a diagnosis at the time of autopsy of metastatic carcinomatosis, primary source not found.

When the sections came through we were rather surprised to find that it was not a carcinoma but a fibrosarcoma of a highly malignant type. We were then left with the problem of trying to decide where this fibrosarcoma might have been primary. None of the foci that we found were points where fibrosarcomas are particularly prone to occur. It would certainly be unlikely to be primary either in the bone marrow or adrenals, and primary sarcoma of the meninges is very uncommon, to say the least. The dura being, however, the largest and most conspicuous focus of tumor and a perfectly possible primary site, we finally made that diagnosis. The most extensive metastases were found throughout the bone marrow of the flat bones and vertebrae, which was almost completely replaced by tumor. There was some compensatory hyperplasia in the long bones, where a certain amount of blood formation was still going on.

A PHYSICIAN: Did she have metastases in the brain?

DR. MALLORY: We found no explanation for the paralysis. There were no metastases in the brain, and Dr. Kubik was not able to find any vascular lesions.

A PHYSICIAN: What were the lesions in the lungs?

DR. MALLORY: A combination of bronchopneumonia and metastatic tumor, almost entirely within the blood vessels. Many of the pulmonary arteries were filled with thrombi of tumor cells, but no tumor was found in the parenchyma of the lungs.

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OPTOMETRY IS NOT MEDICINE!

Newspaper reports on a recent decision (printed elsewhere in this issue of the *Journal*) of the Supreme Judicial Court to the effect that the Court has declared that optometry is not the practice of medicine have aroused fears that chiropody or chiropractic or ophthalmology or surgery might be declared not to be the practice of medicine just because a special board of registration had been created. Medicine might become only what was left after all the special methods of healing had been separated from it. Such fears are unfounded for, as the opinion says: "the question now before us is purely one of statutory construction."

In the case of McMurdo vs. Getter, to which reference was made in a recent issue of the *Journal* (*218*: 402-404, 1938), the professional status of optometry was regarded as established, and the later opinion comments on the earlier one as follows. "Indeed the whole line of reasoning in that case, which was decided after careful consideration and wide search into the authorities, is inconsistent with the theory that optometry as now defined in this Commonwealth has been and now is a part of the practice of medicine. That case proceeds upon the theory that optometry acquired its present professional status solely by force of the statutes expressly relating to that particular subject and not as a branch of the practice of medicine." The opinion closes with these words: "We are compelled to conclude that the petitioner, although he was a registered physician, was not carrying on the practice of medicine within the meaning of General Laws (Tercentenary Edition), Chapter 112, Section 2. The decree must be reversed and in accordance with the stipulation of the parties, a decree is to be entered reversing the decision of the board. Ordered accordingly."

The popular interpretation of this opinion, namely that optometry is not the practice of medicine, is so discrepant from the judgment of common sense that the qualifications stated in the opinion must be scrutinized carefully. Any reasonable definition of the practice of medicine will include what the optometrist claims he may properly do: for example, "make a diagnosis of brain tumor," as an optometrist said at a public hearing. This claim is supported by the present statute.

The critical point lies in the last sentence of General Laws (Tercentenary Edition), Chapter 112, Section 73, which reads as follows: "But nothing herein contained shall prevent any such physician or surgeon from taking an examination and receiving a certificate of registration under section sixty eight, nor shall this and the seven preceding sections authorize any person to administer drugs in any form, to practice or claim to practice medicine or surgery in any sense, or to use any title or appellation intended or calculated to indicate the practice of medicine or surgery." Ergo, optometry is not the practice of medicine or surgery, *in any sense*.

The opinion naturally follows the statute, but the statute is repugnant to common sense and not

in accordance with the facts of the generally accepted practice of medicine and optometry in Massachusetts. If the phrase had read "in any other sense," it would have been an approximation to a more accurate description of the facts.

What is needed is a clarification of the statutory meaning of the practice of medicine: that is, a definition of the practice of medicine, and of the scope of the subsidiary branches of the healing art for the practice of which special licenses are required by law. To this clarification the Massachusetts Medical Society may well direct its efforts.

NEWER FORMS OF TREATMENT FOR MENTAL DISEASE

Reports are gradually appearing in regard to the newer forms of treatment for certain types of mental disease. For dementia praecox, insulin treatment and the use of metrazol have both been employed for over a year. The *Annual Report* from the New York Hospital, Westchester Division, formerly known as the Bloomingdale Hospital, emphasizes the importance of these methods of therapy as well as reminds us that other methods previously used are not to be discarded. With insulin the staff members believe that none of their patients so treated can be considered as recovered, in so far as the patient is permanently cured of his mental symptoms. A number of their patients were greatly improved, but for comparatively brief periods of time. Often a patient, who showed marked recovery and was able to leave the hospital, returned a few weeks later in a condition not dissimilar to that which he had shown in the past. To be sure, the hospital treated cases which were not early and better results might have been obtained had their series contained patients with symptoms of only six months', or less, duration. It is this early group, however, that also shows the best recovery rate from nonspecific therapy. A study of the results in 500 cases admitted to the hospital since 1935 and subsequently discharged as improved or cured is now being made. Although this report is not yet available, from previous studies it has been found that about a third of the patients so discharged are able to make a social adjustment sufficient to live outside the hospital. This percentage is probably higher than that which would be found in state hospitals, for a private hospital of this type has a selected group of patients to treat.

It is interesting to note that, up to the end of 1937, the New York Hospital, Westchester Division, had not attempted metrazol treatment to produce convulsions in patients with dementia praecox. The staff members believed that the likelihood of injury to patients was such that the risk was not justified. Other hospitals, however, have used this interesting form of treatment, and many psychiatrists believe that the results are as good as, or even better than, those with insulin. It is too early to judge the final results.

Another form of treatment concerns the use of estrogenic hormone in involutional melancholia. One half the patients so treated at this hospital responded very well to the drug and showed a noticeable decrease in feelings of tension, agitation, restlessness and sleeplessness. It would seem that the use of this hormone in involutional melancholia is of distinct value.

Finally, Sodium-Amytal narcosis was used in patients who were disturbed, destructive and assaultive. The drug was administered usually by rectum, over a period of ten days, in doses starting from 0.6 gm. and gradually increasing to a maximum of 5.0 gm. per day. Five of eight patients were greatly benefited by this treatment both physically and mentally, and the report states that this form of therapy is considered to be helpful for selected patients.

In general, it should be noted that great activity is now being shown by physicians in charge of psychiatric patients in the use of newer forms of therapy and that the outlook for cure, or at least amelioration, of some types of mental disease is perhaps brighter than at any time in the past. There is a noteworthy conservatism, however, about the reports of the efficacy of these methods; such an attitude is entirely commendable when one looks back into the history of psychiatric therapy.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

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CASE HISTORY No. 83. COMPLETE PLACENTA PREVIA

Mrs. A. L. M., a forty-year-old para III, was admitted to the hospital on the evening of February 18, 1929. She was about twenty-nine to thirty weeks pregnant, and earlier on the same evening, without pain or other warning, she had had a considerable amount of bright-red vaginal bleeding. Her doctor had taken her at once to the hospital.

The patient had had no serious illness, and her family history was not important. Catamenia had begun at twelve, were fairly regular with a twenty-eight-day cycle and lasted four or five days. Her previous pregnancies had been uneventful. The last period had been July 28, confinement being expected early in May. This pregnancy, prior to the evening on which she was first seen, had been uncomplicated.

On physical examination the patient was well nourished. Her skin and mucous membranes showed no pallor. Nothing abnormal was found in heart or lungs. The pulse was 100, the temperature 98.6°F., and the blood pressure 120 systolic, 80 diastolic. The abdomen was rounded, the fundus being slightly above the umbilicus; there was no tenderness on palpation. Fetal movements could be plainly seen and felt. There was a bright-red stain on the vaginal pad.

The patient was put on a delivery table, shaved and prepared vaginally, catheterized, and draped with sterile sheets. A sterile bagging set and packing strips were at hand. Without anesthetic, a vaginal examination was made. The perineum gave good support, the cervix was extremely soft, and the external os admitted a finger tip. A blood-clot cast of the vagina and cervical canal was removed without fresh bleeding's occurring. The internal os did not admit a finger tip, but on palpating through the anterior vaginal wall there seemed to be a soft mass, anterior to the cervix, between the presenting part and the examining finger. A bivalve vaginal speculum was inserted which gave a good view of the cervix. No polyp or bleeding erosion was seen, and only the faintest trickle of blood issued from the os.

The patient was returned to bed; her blood was found to be compatible with that of her husband. She was kept in bed under close observation from then on.

On March 18 a second vaginal examination was made. This time, with a finger pushed through the internal os, the fetal head could be felt, and no placenta was found around the margin.

Since the patient and her family were insistent and since no definite evidence of a placenta previa had been obtained, she was allowed to go home, though she was still kept in bed and under close supervision. The urine and blood pressure remained normal during this time; the fetus continued active; and the patient had no untoward signs or symptoms.

On May 1, seventy-two days after the initial bleeding, she awoke early and found that she had had some vaginal bleeding without pain. She was seen at once and taken by ambulance to the hospital. Examination at that time showed her to be approximately at term. Vaginally, the placenta could be felt across the internal os, and the presenting part could not be felt. A donor for a possible transfusion was secured, and when satisfactory cross-typing was completed, the patient was prepared for operation. Under nitrous-oxide, oxygen and ether anesthesia, a low classical cesarean section was done. A baby weighing 6 lb., 9 oz., was extracted and cried well at once. The placenta was found to be attached low on the uterine wall, its edge being completely across the os. This, with the membranes, was removed entire, and the uterine and abdominal wounds closed. There was no excessive bleeding. The mother made an uncomplicated recovery, and she and the baby went home well on the sixteenth postpartum day.

Comment. This case illustrates the generally recognized method of treating complete placenta previas when the patients bleed at term. The examination when the patient entered the hospital might well have been postponed, as the patient was not actively bleeding. Intelligent procrastination — keeping the patient in bed in the hospital, for the sake of the baby, unless further bleeding started — might have been a little wiser. However, so long as the internal os was not invaded, no possibility of starting up bleeding existed. The second examination when the finger was pushed through the internal os and no placenta felt ruled out, at the moment, the existence of a complete previa. At that stage of pregnancy the only placenta previa that can be felt by inserting the finger is the centrally implanted one. It is not an unusual occurrence to examine a patient at thirty or thirty-six weeks who has bled and find no placenta at that time lying over the cervix and at a subsequent bleeding to find a placenta previa that is complete. This is due entirely to the taking up of the cervix

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

and the thinning of the lower segment, events which allow the placenta to become complete because of the change in the lower segment itself. The examination when this patient again bled established the diagnosis of complete placenta previa, and as she was not in labor and the baby was viable, cesarean section was undoubtedly the conservative procedure.

DEATHS

BARRÉ — JOSEPH A. BARRÉ, M.D., of Fall River, died July 25. He was in his seventy-first year.

Dr. Barré graduated from the College of Physicians and Surgeons of Baltimore in 1892. He was a member of the Massachusetts Medical Society and was at one time a member of the Police Commission and the Board of Health in Fall River.

A widow, a daughter, two sons and five sisters survive him.

NUTE — ALBERT J. NUTE, M.D., of 316 Hyde Park Avenue, Jamaica Plain, died July 25. He was in his sixty-third year.

Born in Boston, he attended Boston English High School, the University of Maine and Harvard College and was graduated from the Harvard Medical School in 1902.

Dr. Nute entered the United States Public Health Service in 1905 and acted as assistant surgeon. He was a former member of the Massachusetts Medical Society, a member of the Naval Lodge of Masons in Kittery, Maine, and of the Medical Veterans of the World War.

A sister-in-law and a cousin survive him.

MISCELLANY

COURT DECISION RELATIVE TO THE PRACTICE OF OPTOMETRY

The following decision is reprinted from "Advanced Sheets" (1938), pages 1063 through 1069:

OPINIONS OF THE SUPREME JUDICIAL COURT

BARNEY EDWARD SACHS *vs.* BOARD OF REGISTRATION IN MEDICINE

Present: FIELD, LUMMUS, QUA, & DOLAN, JJ.

Appeal by the petitioner from a decree entered by order of *Donahue*, J., dismissing a petition in the Supreme Judicial Court for the county of Suffolk.

QUA, J. This is a petition under G. L. (Ter. Ed.) c. 112, § 64, by a physician whose registration has been canceled by the board of registration in medicine. The board found that the petitioner "was acting as principal or assistant in carrying on the practice of medicine with an unlicensed person." In canceling the petitioner's registration, the board purported to act under the authority of G. L. (Ter. Ed.) c. 112, § 2.

The case was heard upon a "Stipulation of Facts," at the end of which is the following: "The sole question raised is whether the petitioner in performing the acts herein described is practicing medicine. It is agreed that if it appears that the petitioner was practicing medicine, the decision of the board is correct and its findings and order should be upheld. It is further agreed that if the Court finds that the petitioner was not practicing medicine, then the decision of the board is to be reversed and the license and registration be ordered restored to the petitioner." Thus it becomes our duty to decide the single question whether upon the facts agreed the petitioner was "carrying on . . . the practice of medicine" within the meaning of those words in G. L. (Ter. Ed.) c. 112, § 2.

The substantive facts upon which this question is to be decided are these: The petitioner and one Getter, "a lay person," were associated in the optical business known as "State Opticians" at Worcester. The capital was supplied by Getter and one Morris Sachs, a brother of the petitioner. Getter held the lease of the premises. He usually assisted the customer in selecting the style of frame after the customer's eyes had been examined. He and the petitioner drew weekly equal amounts from the income. Morris Sachs was regularly employed in Boston, but occasionally "assisted around the place" in Worcester. He drew no money. Business was obtained through newspaper advertising in the name of "State Opticians." Glasses designed to correct "the defective vision of the purchaser" were furnished complete for a single price. There was no special charge for the examination or the prescription. After the examination the lenses were ground as prescribed, fitted to the frame selected, and adapted by an employee to the eyes of the customer. The firm "State Opticians" owned all optical instruments and equipment and paid all expenses. A part of the space upon the premises was partitioned for the office of the petitioner. His name followed by "M.D." was on the door, and his certificate of registration was displayed in his office. Customers were referred for examination to the petitioner. He diagnosed any optical defect, deficiency or deformity and, if necessary, prescribed the lenses or prisms for its correction or relief. He used no drugs and performed no surgical operations. He did only such acts as are included in the definition of optometry contained in G. L. (Ter. Ed.) c. 112, § 66, as amended by St. 1934, c. 339, § 2.

The practice of medicine as ordinarily understood covers a wide field. It does not necessarily involve the use of drugs. *Commonwealth* v. *Jewelle*, 199 Mass. 558. It commonly includes bone setting, *Commonwealth* v. *Dragon*, 239 Mass. 549, midwifery, *Commonwealth* v. *Porn*, 196 Mass. 326, chiropractic, *Commonwealth* v. *Zimmerman*, 221 Mass. 184, *Whipple* v. *Grandchamp*, 261 Mass. 40, 44, and by express statutory provision, osteopathy. G. L. (Ter. Ed.) c. 112, § 10. The work of an "eye specialist," *Commonwealth* v. *St. Pierre*, 175 Mass. 48, 51, and of an ophthalmologist, *Commonwealth* v. *Houtenbrink*, 235 Mass. 320, may also constitute the practice of medicine. In this connection, however, it must be remembered that by etymology, by common usage and by judicial definition ophthalmology is a word of much broader signification than optometry. Thus in the recent case of *New Jersey State Board of Optometrists* v *S. S. Kresge Co.* 113 N. J. L. 287, 294, it is said of the distinction between the work of oculists and ophthalmologists on the one hand and that of optometrists on the other that "The first has relation to the practice of medicine and surgery in the treatment of diseases of the eye, and the second to the measurement of the powers of vision, and the adaptation of lenses for the aid thereof."

We have no disposition to adopt a narrow or con-

stricted view as to what activities are comprised within the practice of medicine, particularly in those aspects which bear upon the maintenance of the public health and upon the protection and security of those who resort for aid to persons holding themselves out as possessing peculiar knowledge and skill in the treatment of disease. Nevertheless, as stated at the outset, the question now before us is purely one of statutory construction. As to such a question reference should first be had to the statutes themselves for such assistance as may be derived from their language, their chronology, and their form and structure with relation to each other. See *Commonwealth* v. *S. S. Kresge Co.* 267 Mass. 145, 148.

St. 1894, c. 458, which established the board of registration in medicine and first required an examination and certificate as a condition of the right to practise, contained no definition of the practice of medicine and permitted the employment of any "method of healing" by unregistered persons who did not hold themselves out as physicians or surgeons. See now G. L. (Ter. Ed.) c. 112, §§ 1-12A and §§ 61-65. When, however, by St. 1912, c. 700, the Legislature first regulated the practice of optometry, it specifically defined that practice as "the employment of any method or means other than the use of drugs for the measurement of the powers of vision and the adaptation of lenses for the aid thereof." § 1. This act was not an amendment of the law governing the practice of medicine. It set up an entirely new board for the examination and registration of applicants. §§ 2-5. It was not in form a new or different method of regulating a branch of the practice of medicine. Its form and substance were such as would naturally be chosen as appropriate to a new subject not regarded as already covered by statute. Moreover, although the act expressly exempted from its provisions physicians and surgeons "entitled to practise medicine in the commonwealth," it allowed them to be examined and registered as optometrists, and it contained this significant provision, "nor shall this act be construed to authorize any person to administer drugs in any form, to practise or claim to practise medicine or surgery in any sense, or to use any title or appellation intended or calculated to indicate the practice of medicine or surgery." § 10. It is difficult to believe that the Legislature regarded optometry, carefully defined in this statute, as a part of the practice of medicine under previous statutes, when in authorizing persons to practise optometry the Legislature itself declared that it did not authorize them to practise medicine "in any sense." The exception of physicians from the operation of the new act would seem to have been due, not to the belief that "optometry" as there defined was "medicine," but to the belief that there was no necessity for regulating qualified physicians who practised in the field of optometry. The act contains in the same sentence a similar exception of persons who merely sell glasses on prescription or as merchandise. § 10. The respondents argue that the petitioner is engaged in practice solely by virtue of his registration as a physician. This is true only in an indirect or qualified sense. An accurate statement would be, we think, that the petitioner may practise optometry, because when the Legislature undertook to regulate optometry it excluded registered physicians from the scope of the act, thereby leaving them still free to practise as all persons had been before the passage of the act.

Subsequent amendments to the act of 1912 do not seem to have affected its construction so far as concerns the question here considered. See now G. L. (Ter. Ed.) c. 112, §§ 66-73A, as amended by St. 1934, c. 339. The definition of optometry has been somewhat particularized so that it now expressly includes "the diagnosis of any optical defect, deficiency or deformity of the human eye, or visual or muscular anomaly of the visual system," G. L. (Ter. Ed.) c. 112, § 66, as amended by St. 1934, c. 339, § 2, but it does not include the general treatment of diseases of the eye, and the provision differentiating the practice of "optometry" from the practice of "medicine or surgery in any sense" is still retained. G. L. (Ter. Ed.) c. 112, § 73, as amended by St. 1934, c. 339, § 2. The penalties for violations of law differ with respect to the two professions. Compare G. L. (Ter. Ed.) c. 112, § 6, with G. L. (Ter. Ed.) c. 112, § 72A, as inserted by St. 1934, c. 339, § 2.

Some assistance may be found in the manner in which the Legislature has dealt with other professions or occupations more or less analogous to the practice of medicine. Thus pharmacy and dentistry are each under the jurisdiction of a separate board, although physicians are in some specified respects exempted from the operation of the laws applicable to those professions. G. L. (Ter. Ed.) c. 112, §§ 24 and 35, as amended, §§ 43 and 53. So also nursing and veterinary medicine are under separate boards. G. L. (Ter. Ed.) c. 112, §§ 74, 54, and 55 as amended. But osteopathy, which is a method of curing human disease, is under the board of registration in medicine, G. L. (Ter. Ed.) c. 112, § 10, and chiropody, the statutory definition of which expressly includes medical and surgical treatment, was likewise under that board until 1937. St. 1917, c. 202, § 3. St. 1937, c. 425, §§ 1, 2. As to none of these occupations is there any provision comparable to that hereinbefore quoted which bars the registered optometrist who is not a physician from practising "medicine or surgery in any sense."

Other considerations of a more general nature tend toward the conclusion that one who practises optometry exclusively is not commonly to be treated as practising medicine. Optometry in its origin and nature is more akin to the physical science of optics than to the science of medicine. Its emphasis today is upon supplying physical means to aid the bodily powers rather than upon the cure of disease. In the recent case of *McMurdo* v. *Getter*, Mass. Adv. Sh. (1937) 1355, 1360, decided after the entry of the final decree in this case, it is said that "Undoubtedly the fitting and sale of eyeglasses began as a trade and not as a profession." Indeed the whole line of reasoning in that case, which was decided after careful consideration and wide search into the authorities, is inconsistent with the theory that optometry as now defined in this Commonwealth has been and now is a part of the practice of medicine. That case proceeds upon the theory that optometry acquired its present professional status solely by force of the statutes expressly relating to that particular subject and not as a branch of the practice of medicine.

In other jurisdictions, although the varying terms of the statutes may affect the result, it has been widely held, stated, or implied that practising optometry, without at any point overstepping the bounds which define that word, is not practising medicine. *Martin* v. *Baldy*, 249 Penn. St. 253. *People* v. *Smith*, 208 Ill. 31. *People* v. *Griffith*, 280 Ill. 18. *In re Rust*, 181 Cal. 73. *Saunders* v. *Swann*, 155 Tenn. 310. *Dvorine* v. *Castelberg Jewelry Corp.* 170 Md. 661. *State* v. *Rust*, 119 Wash.

480. *Bennett* v. *Indiana State Board of Registration & Examination in Optometry,* 7 N. E. Rep. (2d) 977, 981 (Ind.). *Funk Jewelry Co.* v. *Arizona,* 46 Ariz. 348, 354. *State* v. *Buhl Optical Co.* 131 Ohio St. 217, 221. *McNaughton* v. *Johnson,* 242 U. S. 344. In *Georgia Board of Optometry* v. *Friedman,* 183 Ga. 669, many recent cases are collected illustrating the modern recognition of optometry as an independent profession or calling. And see cases collected in 22 Am. L. R. 1173. Compare, however, *State* v. *Corriveau,* 131 Maine, 79; *Swanz* v. *Clark,* 71 Mont. 385; *State* v. *Yegge,* 19 S. D. 234.

We are compelled to conclude that the petitioner, although he was a registered physician, was not carrying the practice of medicine within the meaning of G. L. (Ter. Ed.) c. 112, § 2. The decree must be reversed, and in accordance with the stipulation of the parties a decree is to be entered reversing the decision of the board.

Ordered accordingly.

TUBERCULOSIS EXHIBITS

A new exhibit on the importance of x-ray in the diagnosis of tuberculosis is available to medical groups at no cost, subject to its itinerary. This exhibit, shown at the American Medical Association meeting in San Francisco, can be secured through the Massachusetts Tuberculosis League, 1148 Little Building, Boston. Thirty running feet of wall space is desirable, but the exhibit can be shown on tables.

A smaller loan exhibit showing the technic of testing with purefied protein derivative (P.P.D.) is also available. It consists of six panels, each 24 by 36 inches, and comes in a case, weighing in all 110 lb.

CORRESPONDENCE

A CORRECTION

To the Editor: At the request of Dr. Nels Nelson, we wish to correct a misstatement in our article "Sulfanilamide in the Treatment of Gonorrhea in the Female" published in the *New England Journal of Medicine* (*218:* 1092-1094, 1938).

The drug used was not furnished directly by the Massachusetts Department of Public Health but was purchased through a fund given by the State for treating the gonorrheal patients at the Boston City Hospital Clinic.

BURTON C. GRODBERG, M.D.,
EDMUND L. CAREY, M.D.

Boston City Hospital,
Boston.

RESULTS OF STATE BOARD EXAMINATION

To the Editor: The following tabulations are based on the results of the March, 1938, examination given by the Massachusetts Board of Registration in Medicine.

STEPHEN RUSHMORE, M.D., *Secretary.*

State House,
Boston.

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The first table gives the name of the school, the number of candidates from each school, the number who passed, the number who failed and the percentage of the graduates who passed. The order in which the schools are listed is merely the order in which numbers for examination were assigned to the first candidate from the school who made application for examination.

LOCATION OF SCHOOL	TOTAL CANDIDATES	NUMBER PASSED	NUMBER FAILED	PERCENTAGE PASSED
Kansas City University of Physicians and Surgeons	20	1	19	5
University of Ghent	1	0	1	0
Philadelphia College of Osteopathy	3	0	3	0
Middlesex University	79	9	70	11
College of Physicians and Surgeons (Boston)	11	4	7	36
Mid-West Medical College	10	2	8	20
Massachusetts College of Osteopathy	11	1	10	9
University of Lausanne	1	0	1	0
Tufts College	8	6	2	75
New York Homeopathic Medical College	1	1	0	100
Des Moines College of Osteopathy	1	0	1	0
Kirksville College of Osteopathy	5	1	4	20
University of Maryland	3	3	0	100
Royal Colleges (Edinburgh)	3	1	2	33
University of Geneva	1	0	1	0
University of Munich	1	0	1	0
Harvard University	8	8	0	100
McGill University	3	3	0	100
University of Leipzig	1	1	0	100
University of Cologne	1	1	0	100
Boston University	3	2	1	66
Georgetown University	2	2	0	100
Baylor University	1	1	0	100
University of Naples	1	0	1	0
University of Minnesota	1	1	0	100
Northwestern University	1	1	0	100
Vanderbilt University	3	3	0	100
University of Indiana	1	1	0	100
University of Würzburg	1	0	1	0
Rush Medical College	1	1	0	100
Johns Hopkins University	1	1	0	100
University of Michigan	3	3	0	100
Columbia University	1	1	0	100
University of Chicago	1	1	0	100
University of Berlin	1	1	0	100

The second table indicates the grouping of the schools from which the candidate came, in relation to the results of the examination and to the number of times the candidate had been examined.

	APPROVED SCHOOLS	NON-APPROVED SCHOOLS	OSTEOPATHIC SCHOOLS
FIRST EXAMINATION			
Number Examined	43	18	1
Number Passed	40	5	1
Number Failed	3	13	0
SECOND OR THIRD EXAMINATION			
Number Examined	10	74	13
Number Passed	4	9	1
Number Failed	6	65	12

FOURTH OR MORE EXAMINATION			
Number Examined	1	28	6
Number Passed	0	1	0
Number Failed	1	27	6
TOTALS			
Number Examined	54	120	20
Number Passed	44	15	2
Number Failed	10	105	18

RECAPITULATION

Number Examined	194
Number Passed	61
Number Failed	133
Percentage Passed	31

NOTICES

TUMOR CLINIC, BOSTON DISPENSARY

Each Tuesday and Friday morning from ten to twelve-thirty there is a meeting of the Tumor Clinic of the Boston Dispensary, a unit of the New England Medical Center. All kinds of tumors are seen, discussed, and when indicated, treated with radium and high-voltage x-ray.

Physicians are welcome to visit this clinic and bring patients to the clinic for diagnosis.

CONGRESS OF THE INTERNATIONAL SOCIETY OF SURGERY

The eleventh congress of the International Society of Surgery which, as previously announced, was to have been held in Vienna on September 19 to 22, under the auspices of the German government and with the cooperation of the German Surgical Society, has, for reasons not yet explained, been made impossible in Vienna. Fortunately, the transfer of the congress from Vienna to Brussels, which was promptly effected early in July by the Executive Bureau of the International Society of Surgery, has proved of great advantage in assuring a greater success of the congress under the liberal patronage of the Belgian government and the enthusiastic support of the Belgian medical profession. There will be no change of date. According to the latest information received from the secretary-general, Dr. Leopold Mayer, the scientific program will be carried out in Brussels in all its details as previously announced for Vienna. In fact, many of the reports assigned to the eminent specialists who are to open the discussions on the official questions selected for debate have been printed, and others in press will be ready for distribution in advance of the meeting.

The prospects of the elaborate Mediterranean cruise, which has been planned by the Belgian branch of the French Navigation Company, under the direction of the International Bureau at Brussels, will be carried out in full, differing only from the itinerary originally planned for the Vienna congress by omitting the stops at Budapest and Vienna, and substituting the return to Brussels by rail via Venice, Milan and Basel, in time to arrive in Brussels on September 17 or 18. The cruise will begin in Marseilles on September 4, on board the French packet *Le Providence* (15,000 tons) and will continue along the north Mediterranean to the Black Sea, with stops at Naples, Athens, Istanbul (Constantinople), Odessa, Gostanza and Bucharest, to return to Brussels by the inland route.

Ample provision for sightseeing and entertainment has been assured by the appointment of active medical committees in each of the stopping places along the route, besides the assurance of success given by the long experience of the Secretary-General, who will personally supervise the management of the cruise in its scientific and touristic aspects. During his absence, the secretarial office in Brussels will be occupied by five subsecretaries, Drs. Neumann, Appelmans, Beckers, Dejardin and Spehl, who will attend to all the business of the congress until Dr. Mayer's return.

It is probable that the transfer of the congress from Vienna to Brussels will increase the popularity of the Mediterranean cruise, and that the demand for reservations on shipboard will be in excess of the available space. It is important, therefore, that all the fellows of the Society, and others who may desire to join the Mediterranean cruise, should apply at once to Dr. Leopold Mayer, 72 Rue de la Loi, Brussels, or directly to the Belgian office of the Compagnies Françaises de Navigation (29 Boulevard Adolph Max, Brussels).

At the present time the American Committee has given authority to the American Express Company to set up group travel direct to Brussels as well as to outline several trips which will bring the fellows to Marseilles by September 4, where, as it is described above, the French packet *Le Providence* will start on its Mediterranean cruise to the Black Sea.

Membership in the International Society of Surgery and in the congress alone will entitle one to reduced rates and privileges over practically all the European railways and other means of transportation on presentation of the certificate or card of membership. All the procedures required to secure reduction of fares and other privileges in the different European countries are fully detailed in the circulars recently issued to all the fellows of the Society by the Secretary-General, the Belgian Touristic Agency of the French Navigation Company, and by the American Express Company.

RUDOLPH MATAS, M.D., *President,*
ELLIOTT C. CUTLER, M.D., *Chairman,*
American Committee.

CONFERENCE ON RURAL MEDICINE

As a result of contact with many of the problems of rural medicine, the staff of The Mary Imogene Bassett Hospital, Cooperstown, New York, has undertaken to conduct a Conference on Rural Medicine on October 7 and 8. The purpose of this conference is to define and, if possible, by description and discussion to illuminate some of the problems of rural medicine.

The four general subjects will be: rural morbidity — discussions of this subject will be in part based upon a statistical analysis of upwards of 15,000 hospital admissions in a rural area of New York State; public health programs in rural areas — the topics to be considered will be (a) health-department programs and (b) school health programs; postgraduate medical education in rural areas; and the economics of rural medicine — the subdivisions of this subject will be (a) the rural hospital, (b) the rural physician and (c) the member of a rural community.

The Advisory Committee consists of the following: Dr. Marion A. Blankenhorn, director, Department of Internal Medicine, University of Cincinnati; Dr. Frank G. Boudreau, executive director, The Milbank Fund; Dan D. Casement, Juniata Farm, Manhattan, Kansas; Dr. Michael M. Davis, chairman, Committee on Research in Medical Economics; Dr. Louis I. Dublin, third vice-president and statistician, Metropolitan Life Insurance Company; Dr. Thomas P. Farmer, member of the council, Medical So-

ciety of New York State; Dr. Edward S. Godfrey, Jr., commissioner of health, State of New York Department of Health; Dr. H. S. Mustard, director, Department of Preventive Medicine, New York University College of Medicine; Dr. Willard C. Rappleye, dean, College of Physicians and Surgeons, Columbia University; Prof. Lowell J. Reed, director, Department of Biostatistics, and dean, School of Hygiene and Public Health, Johns Hopkins University; Dr. Ray Lyman Wilbur, president, Stamford University; Owen D. Young, chairman of the board, General Electric Company.

GRADUATE FORTNIGHT OF THE NEW YORK ACADEMY OF MEDICINE

The eleventh annual Graduate Fortnight of the New York Academy of Medicine will be held in New York City from October 24 through November 4. The general topic is "Diseases of the Blood and Blood-Forming Organs."

As usual the clinical sessions will be held during the day at the various hospitals. During the evening, talks on different aspects of the general topic will be given at the Academy by men who are experts along such lines. The subtopics to be discussed are as follows: "Macrocytic Anemias," "Anemias Due to Iron Deficiency," "Refractory Group of Anemias and the Granulocytopenias," "Blood Formation and Destruction: Hematohistology," "Diagnostic Procedures," "The Leukemias and Leukemoid States," "Malignant Diseases of the Lymph Nodes and Bone Marrow," "The Polycythemias," "The Hemorrhagic Diseases," and "Diseases of the Spleen and Therapy." There will be an extensive scientific exhibit at the Academy, and fresh pathological material will be demonstrated during the evening sessions.

Registration is limited to the medical profession, and a registration fee of three dollars is required from those who are not members of the Academy. Further details can be obtained by writing to the New York Academy of Medicine, 2 East 103 Street, New York City.

AMERICAN ASSOCIATION OF RAILWAY SURGEONS

Never before have problems confronting railway surgeons been so grave economically, or technic and treatment procedures of such import as they are this year.

The twenty-third annual meeting of the American Association of Railway Surgeons will be held at the Palmer House, Chicago, September 19 to 23.

This association includes members in practically every railroad company in the United States, as well as the separate group organizations, embracing railroad surgeons of the New York Central System; Southern Railway; Atlantic and West Point Railroad; Western Railway of Alabama; Illinois Central System; Chicago, Milwaukee, St. Paul and Pacific Railroad; Rock Island Lines; Chicago, Burlington and Quincy Railroad; Chicago and Northwestern Railroad; the Georgia Railway; and others.

An extremely interesting and highly profitable program has been arranged, and all physicians and surgeons are invited to attend the sessions of this meeting as guests of the organization. There will be no registration fee to non-member guests. The complete program and information regarding the meeting and the exhibits may be secured by addressing Mr. A. G. Park, American Association of Railway Surgeons, Palmer House, Chicago, Illinois.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, AUGUST 8

TUESDAY, AUGUST 9

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

FRIDAY, AUGUST 12

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

SATURDAY, AUGUST 13

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Marshall N. Fulton.

*Open to the medical profession.

SEPTEMBER 12-14 — American Association for the Study of Goiter. Page 545, issue of March 24, and page 145, issue of July 28.

SEPTEMBER 12-15 — American Congress of Physical Therapy. Page 946, issue of June 2.

SEPTEMBER 19-22 — Congress of the International Society of Surgery. Page 180.

SEPTEMBER 19-23 — American Association of Railway Surgeons. Notice elsewhere on this page.

SEPTEMBER 26 and 27 — Symposium on Occupational Disease. Page 145, issue of July 28.

OCTOBER 7 and 8 — Conference on Rural Medicine. Page 180.

OCTOBER 8 and NOVEMBER 15 — American Board of Ophthalmology. Page 282, issue of February 10.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

OCTOBER 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

OCTOBER 24 - NOVEMBER 4 — Graduate Fortnight of the New York Academy of Medicine. Notice elsewhere on this page.

NOVEMBER 5 — American Board of Obstetrics and Gynecology. Page 145, issue of July 28.

MARCH 27-31, 1939 — American College of Physicians. Page 36, issue of July 7.

BOOK REVIEWS

Management of the Sick Infant and Child. Langley Porter and William E. Carter. Fifth revised edition. 874 pp. St. Louis: The C. V. Mosby Company, 1938. $10.00.

This fifth edition of the authors' well-known textbook brings the material presented up to date, at the time of writing.

The interesting feature of presenting the material from two methods of approach — first, the presenting symptoms, and secondly, the bodily system affected — makes it possible to refer to the text for reference even though the diagnosis of an individual case under consideration may not be established. There is, of necessity, a repetition in the two parts of the book, but this does not detract from its value.

The use of heavy type throughout the text serves to accentuate the conditions mentioned but detracts from the appearance of the text and also somewhat from its readability.

In the second part of the book, which qualifies diseases according to the bodily system affected, it would be clearer if they were presented under the usual headings, such as "Diagnosis" and "Treatment," and if these headings were more clearly differentiated from the text.

The addition at the end of the book of methods employed in diagnosis and therapeusis should serve as a ready means of reference to those carrying out such procedures.

The field of pediatric practice is not completely covered, and some subjects are referred to very briefly, but in general the common conditions encountered are adequately presented. Some difference of opinion might arise with reference to recommendations for treatment, and sometimes the statements concerning specific conditions are

general and somewhat vague. The book, as a whole, is a useful addition to the library of any physician who is dealing with the diseases of infants and children.

Essentials of Psychiatry. George W. Henry. Third edition. 465 pp. Baltimore: The Williams & Wilkins Company, 1938. $5.00.

This book, now in its third edition, has become one of the standard books of American medicine. Two revisions have been called for since the book was originally issued in 1925. Dr. Henry has kept the volume up to date and the present edition reflects the progress of psychiatry during the last five or six years. In addition, the older material has been revised and the author has added a few notes on the history of his subject. One would wish that he had expanded this part of his book and even printed in small type a page or two of historical introduction to each chapter. This would add greatly to the value of the book.

This book should be considered as an introduction to psychiatry and is an excellent text to put into the hands of third-year medical students or practitioners who wish to have a broad understanding of a most complicated branch of medicine. One should not expect to find, however, details of modern forms of treatment, for there is only a brief chapter dealing with the principles of treatment. For information regarding the technic or an evaluation of the newer forms of surgery of the frontal lobes, shock treatment with insulin or metrazol and other modern devices, one would have to look elsewhere. This is not a criticism of the book, for the author has definitely kept within the scope of his title, *Essentials of Psychiatry*.

Les Eléménts du Pronostic dans les Maladies Aiguës: Notes de pratique. A.-B. Marfan. 75 pp. Paris: Masson et Cie, 1938. 20 Fr. fr.

This small brochure consists of a series of articles published during 1937 in the *Journal des Practiciens*. It is mainly a rehash of all the old, commonly recognized, clinical features of infectious diseases that have been used for bedside prognosis by our forefathers, such as a detailed description of the different appearances of the tongue in severe cases of typhoid and pneumonia. Some more recent items are introduced, such as the prognostic significance of serum treatment in diphtheria and of sulfanilamide therapy in meningococcal meningitis.

The acute infectious diseases considered are: typhoid fever, primary lobar pneumonia, bronchopneumonia, diphtheria, scarlet fever, acute endocarditis and pericarditis — including the rheumatic and the purulent types — and meningococcal meningitis. Mention is made of the more benign course of typhoid fever and of diphtheria in individuals who have previously been vaccinated against these infections. The significance of early and adequate treatment of diphtheria and its influence on the prognosis are stressed, as is the importance of recognizing the malignant form of this disease, which requires huge doses of serum. The author believes that the efficacy of antimeningococcus serum has definitely declined in recent years. The excellent results following the use of sulfanilamide in meningococcal meningitis are stressed. The dosage recommended is 2 gm. daily in four doses to infants and 6 gm. daily, in divided doses, in adults — the drug being given by mouth if possible. In scarlet fever the results of the use of antiserums, of convalescent serum and also of sulfanilamide are considered to be still in doubt. At the end of the section on pneumonia the author mentions promising results in combating pneumococcal infections with the use of a new drug, di(p-acetyl-amino-phenyl)sulfone (1399 F.), recently reported by Fourneau and his co-workers. However, there is nothing concerning specific serum therapy in the treatment of pneumonia — an omission common to most Continental writers. Nor is any mention made of the prognostic significance of the etiologic agent and of the presence of bacteremia in this disease — matters now receiving increasing attention in this country.

Perhaps the greatest defect in this work, as in so many similar ones, is the apparent lack of realization that *prognosis* to be useful must depend on the results of careful analysis of a large body of properly gathered data by good observers, and not on clinical impressions gathered from individual cases.

Medicine for Nurses. W. Gordon Sears. Second edition. 435 pp. Baltimore: William Wood & Company, 1937. $3.25.

This small book covers the usual field of general disease, with simple illustrations and easily found definitions. It is designed as a text for nurses during the course of their study. The first edition in 1935 was so popular that a second edition was called for in 1937. In this new volume, material has been added in regard to the oxygen tent, continuous drip transfusion and similar newer methods of treatment. This book from an English hospital superintendent should prove of value as a textbook.

Surgical Anatomy of the Head and Neck. John F. Barnhill. 921 pp. Baltimore: William Wood & Company, 1937. $20.00.

To the surgeon whose work is basically anatomical, to the surgeon who never ceases to be fascinated by a beautiful anatomical exposure, to the surgeon who is only momentarily disconcerted by a gross anomaly, to the surgeon whose anatomical knowledge permits him occasionally to anticipate, rather than be surprised by, an anomaly, this book is a joy and delight. We read in the preface that the treatise represents years of work — really a challenge to a well-known English otolaryngologist who in the 90's said: "Our American students know less anatomy than those from South Africa."

The first 452 pages of this large quarto volume are devoted to text, the last 460 to anatomical plates. The text is divided into thirty-five periods (chapters). Some of the period captions are: "Thyroid and Parathyroid Glands," "Pneumatic Structures of the Mastoid Process," "Middle Ear," "Petrous Pyramid," "Sphenoidal Sinus," "Ethmoidal Cells," "Frontal Sinus," "Facial and Eighth Nerves," "Maxillary Sinus," "Nose," "Pharynx," "Larynx." Over 20 pages are given to a practical consideration of local anesthesia. The diction is conversationally didactic. We see the teacher instructing his class. The anatomical bases of many operations are clearly shown. A treatise which discusses such embryologic arrests as hair lip, cleft palate, maleruption of molar teeth and thyroglossal cyst, might have appropriately included branchiogenic cysts. The anatomical plates are so instructive it is a pity that they have not been indexed.

The work is a masterful contribution. It is, of course, especially designed to meet the needs of the otolaryngologist but will be useful to the general surgeon, brain specialist, the oral surgeon and the ophthalmologist. The physical qualities of the book are excellent; it is well bound and well printed. The highly calendered paper greatly enhances the beauty and clarity of the plates.